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### ARTERIO-SCLEROSIS: ITS NATURE, CAUSES AND TREATMENT.<sup>1</sup>

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ARTERIO-SCLEROSIS or arterio-capillary fibrosis (for capillary implication must be emphasized) is a pathological condition well known to the general public from its association with high blood pressure or hyperpiesia. Sometimes myocardial degeneration is also connected with it, constituting cardio-vascular sclerosis. Chronic renal degeneration may also be manifest in the form of cirrhotic kidney or chronic interstitial nephritis, less often chronic parenchymatous nephritis. Acute nephritis also may be attended by conspicuously high blood pressure.

Roughly the normal systolic blood pressure of an adult male, as estimated by the sphygmomanometer, is about one hundred and twenty millimetres of mercury. The diastolic blood pressure is about two thirds of the amount, that is eighty millimetres of mercury. As age advances the blood pressure

generally, but not necessarily, rises. Various formulae have been suggested for estimating the safe blood pressure at any age. The simplest formula is: Age plus 100. Another and slightly more conservative one is: Age plus 100, less 5% of the total. In the female sex the systolic blood pressure is about ten millimetres of mercury lower than in the male.

Abnormally high blood pressure may be due either to arterio-sclerosis or vascular spasm or to a combination of both factors. In discriminating between these factors an injection of nitro-glycerine or an inhalation of amyl nitrite may be of the very greatest help. Vascular spasm will be greatly alleviated, whereas chronic structural degeneration will not be. The most pressing danger from arterio-sclerosis and hyperpiesia is cerebral hæmorrhage or apoplexy and the estimation of the blood pressure is not always of very great assistance in foretelling this calamity. If the arterio-sclerosis produces headache, giddiness, mental aberration or epistaxis, cerebral hæmorrhage is always to be feared and yet many patients have systolic blood pressure of well above two hundred millimetres of mercury without manifesting any abnormal signs or symptoms. On the other hand, cerebral hæmorrhage may occur with a blood pressure below 150 millimetres of mercury or even with a normal blood pressure.

<sup>1</sup> Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on October 16, 1924.

Arterio-sclerosis may be classified clinically into three groups (Clifford Allbutt): Hyperpietic, toxic and decreascent or senile arterio-sclerosis.

Hyperpietic arterio-sclerosis is associated with hyperpiesia and is either caused by it or they both result from the action of a pressor stimulant. Of such pressor stimulants adrenalin may be one. The functions of the endocrine suprarenal glands may be vitiated by physical, mental or emotional strain. Mosenthal, however, states that no definite proof has been adduced of a relationship between the adrenals and hyperpiesia. Disturbance of the sodium chloride metabolism has been suggested, but O'Hare and Walker believe that salt plays little or no rôle in the production of arterial hypertension. High protein diets have also been accused and here let me quote from an article by Professor William Russell in *The British Medical Journal* of May 17, 1924. He writes:

I am informed that arterio-sclerosis is alarmingly common in Australia and that it is attributed to excessive flesh eating. The sheep shearers are paid by the hundred fleeces; they work at great speed for hours, they consume three pounds of flesh daily and they all have sclerosed arteries in early life.

Much attention has been directed lately towards the amino-acids or products derived from them by bacterial activity as capable of causing vascular spasm. Tyramine which occurs in ergot, is a substance derived from tyrosin, one of the amino-acids. It has a conspicuous epinephrin-like effect and causes vascular spasm with abnormally raised blood pressure. Exactly the same effect is produced by the metabolite guanidin (which is another amino-acid derivative) with its compounds methyl and dimethyl guanidin.

Toxic arterio-sclerosis may be produced by such toxæmic conditions as gout, syphilis, tuberculosis and pyorrhœa. Here the blood pressure may not be elevated at all. In fact it is notoriously lowered in tuberculosis. In this same group may be placed alcohol, lead, tobacco and even excessive tea and coffee consumption. Tea and coffee contain purin bodies of the methyl-xanthin group. Large intravenous injections of caffeine in animals may cause a slight rise of blood pressure. Caffeine stimulates the vaso-motor centre in the medulla. This would cause a rise of blood pressure but for the fact that caffeine has a direct action on the vessel wall, causing vaso-dilatation. Thus the central action on the circulation is neutralized. If caffeine be administered in very large amounts, cardiac action is weakened and the blood pressure falls to a considerable extent. It is extremely doubtful whether excessive tobacco smoking can actually produce arterio-sclerosis; it may perhaps aggravate the condition. The nicotine content of tobacco varies very greatly and it might be mentioned incidentally here that the cigarette smoker suffers more from carbon monoxide poisoning than from nicotine poisoning. Pharmacologically nicotine exerts a powerful pressor effect; but this is in doses greater than those customarily absorbed by the tobacco smoker. The immediate effect of smoking is a rise of systolic blood pressure (from five to twenty-five millimetres

of mercury). The diastolic blood pressure is less affected. The pulse rate is increased. In a short time, however, the blood pressure falls to or even below the normal. Confirmed smokers generally exhibit a low blood pressure. Tobacco tolerance is diminished as age advances and arterio-sclerotic change increases. Probably also, alcoholic indulgence, even in great excess, cannot produce arterio-sclerosis. Clifford Allbutt says that alcohol is not a cause in eminent domain, but it is a potent ally of any other poison which may be in cooperation with it. In alcoholic intoxication the skin may be flushed from cutaneous vaso-dilatation. This is sometimes accompanied by slight vaso-constriction in the internal organs. Apparently these effects arise from central vaso-motor action. However, it has not yet been decided whether these manifestations are the result of direct stimulation of the centres or arise from a reflex from the stomach. If large quantities of alcohol are consumed, there is a definite fall in arterial tension, through weakening of the cardiac muscle and depression of the vaso-constrictor centres. However, the amount of alcohol necessary to produce a considerable fall of blood pressure is greatly in excess of the quantities therapeutically employed.

In decreascent or senile arterio-sclerosis, due to involutionary or senile changes, the increase in blood pressure may be only moderate. There may even be no increase in the blood pressure (as estimated by the sphygmomanometer) in aged people with conspicuously sclerosed vessels.

Professor Russell holds that the first step in the production of arterio-sclerosis is hypermyotonia, appreciable to the finger applied to the radial and brachial arteries. This is the stage of pre-sclerosis. Continued hypertonia is caused by and is proof of blood impurity. This is to say that there is something in the blood which exerts an irritant or stimulating influence on the arterial wall. This may be absorbed from the alimentary canal or it may be due to the retention of waste products of cell metabolism which ought to have been excreted. The first stage of arterio-sclerosis is thickening of the middle coat, leading to sclerosis or hardening of the arterial wall. This thickening of the *tunica media* is due in the first place to hypertrophy of its muscular fibres. It is altogether different from atheroma. In the second stage fibrous tissue partly replaces the muscle fibres of the middle coat. Some muscle fibres, however, still persist. In the third stage calcification takes place in the thickened and devitalized portions of the *tunica media*. Infiltration takes place irregularly and even now some muscular fibres persist. Russell's classification is not universally accepted. Other authorities maintain that the earliest stage is one of endothelial proliferation visible in the terminal arterioles. This, indeed, may be preceded by capillary changes. Later, the swollen and proliferated endothelial cells undergo fatty degeneration and occlusion of the lumen results. There is a varying swelling of the vessel wall, giving it a moniliform appearance. MacKenzie has come to the definite conclusion that arterio-sclerosis is secondary to obliteration of the

capillaries. Some have adopted a classification of arterio-sclerosis on histological characters, for example, nodular, diffuse, hyaline, fatty, hyperplastic and senile. In atheroma there is a primary change in the intima—a thickening with a tendency to hyaline, mucoid or fatty degenerations, leading to necrosis or calcification. In the early stages the media and adventitia appear normal. The intima may become "as rough as the skin of a crocodile."

The explanation of the signs and symptoms of arterio-sclerosis is not always obvious. However, the functional efficiency of organs depends on their blood supply. If the blood supply fail, the functional efficiency must be impaired. Arterio-sclerosis definitely interferes with the blood supply and so the manifestations of this disorder are seen in impaired function of the organs, including the brain and central nervous system whose blood supply is defective. All organs have periods of increased and diminished activity and the effects of the impaired blood supply will first be evidenced when the organs are exercised to their full functional capacity (MacKenzie). Patients suffering from cerebral arterio-sclerosis may exhibit giddiness or vertigo. Now vertigo may mean anything from the little swim of atonic cephalic vessels to which neurasthenics are subject on stooping, or the passing giddiness of dyspepsia up to labyrinthine disease or a small apoplexy. Labyrinthine vertigo is generally, but not necessarily, attended with deafness or even with tinnitus (Allbutt). In diagnosing the condition the ophthalmic surgeon can render the greatest assistance. He may find "silver wire" arteries, pipe-stem sheathing of arteries, small white "cotton wool" exudates of fibrin or even small hæmorrhages in the *fundus oculi*. Arterio-sclerotic patients are prone to parietic attacks suggestive of cerebral hæmorrhage. These attacks may be transient, giving rise to the assumption that they are due solely to temporary vascular spasm, a diagnosis most comforting to the patient. In such cases treatment may be highly beneficial. Minute hæmorrhages may be the result of diapedesis alone, without any obvious solution of continuity of the vessel wall. Patients may more or less rapidly recover from them. On the other hand, they may be the precursors of an extensive, final and lethal hæmorrhage, as in ingravescent apoplexy. As regards pain arterio-sclerosis in general is a painless disease. At the same time superficial arteries so affected may be tender. Pain in the neck or occiput is frequently associated with arterio-sclerotic disease. We all know the intense pain associated with *angina pectoris* and *abdominis*. The local pain of arterial embolism may be violent; phlebitis and thrombosis are accompanied by pain and tenderness. Intermittent claudication is also attended by pain.

As regards treatment the first essential is to moderate the quantity of food consumed. Over-eating is pernicious. As regards fluid many conflicting opinions have been held. Some advise strict curtailment of the fluid intake so as not to raise the blood pressure. Others with equal insistence have urged the drinking of large quantities of fluids

in order to flush out the kidneys (provided these be working adequately) and the skin and so effect the elimination of noxious toxins. Red meats can profitably be limited, but not interdicted. Herrick states that hyperglycæmia is a feature in a considerable proportion (10% to 20%) of cases of arterial hypertension. This association is commonest in those who are corpulent and exhibit evidence of arterio-sclerosis. In some cases the determining cause seems to be excessive use of carbo-hydrates and the undue restriction of nitrogenous foods. Protein diet should not be unduly restricted unless there is definite evidence of renal inadequacy. Epstein's revolutionary recommendations as to diet in chronic parenchymatous nephritis with œdema might be mentioned in this connexion. Prolonged hyperglycæmia may raise the blood pressure and produce arterio-sclerosis. As regards purins there is a difference of opinion. It is perhaps the safest plan to curtail them strictly. Sodium chloride also should be greatly restricted.

Exercise is necessary in most cases, but caution must be used against over-exertion. It is quite useless to tell patients to avoid all worry of the mind. Nobody worries from choice, but only because he cannot help it. As regards drug treatment, the vaso-dilators occur to one, for example, nitroglycerine and sodium nitrite which are in the British Pharmacopœia. Erythrol tetra-nitrate has been advocated in doses of 0.015 to 0.18 gramme (quarter to three grains). Its action is more prolonged than that of nitro-glycerine, but not so powerful. Mannitol hexa-nitrate has an even more lasting effect than erythrol tetra-nitrate. Hippurates have also been recommended for the purpose of reducing blood pressure. Ammonium hippurate is given in doses of 0.6 gramme or more. As the benzoates are converted in the body into hippurates by combination with glyco-coll, they might be expected to exert the same effect. It is very doubtful, however, whether it is wise to attempt seriously to reduce the blood pressure in such cases. The high blood pressure may have a very definite purpose to serve in maintaining the circulation and metabolism and any considerable reduction may be fraught with serious consequences. "We must recognize that high blood pressure may be in some respects and for some time a beneficial process compensating some disorder of function, just as Thoma considers arterio-sclerosis to be originally a compensatory mechanism to prevent dilatation at the site of weakness of the muscular media." Small doses of bromides may be very valuable as cerebral sedatives to relieve anxiety. Mercury in the form of blue pill or calomel in fractional doses (for example 0.003 gramme—one twentieth of a grain—of the latter) may act as an intestinal antiseptic without being purgative. Of course, free daily action of the bowels is essential. Finally we have the iodides. Clifford Allbutt states that:

The general faith in the iodides as moderators of high arterial pressure is curiously robust and perennial, notwithstanding the default of any definite evidence of this virtue in them.



He further states that sodium nitrite must not be prescribed together with potassium iodide. I cannot tell why. Cushny writes:

Aneurysm and arterio-sclerosis have often been treated with iodide and improvement is undoubtedly observed in some cases in which there is probably a syphilitic taint; but there seems no reason to suppose that the iodides have any special action on the vessels apart from their action on poorly organized tissue, such as is formed in syphilitic infection, for no change in the heart, pulse or blood pressure can be observed even after prolonged treatment.

Lastly high-frequency currents have been used. Probably they have no lasting effects as regards lowering the blood pressure in patients with a constantly raised blood pressure. At the same time their use may alleviate such symptoms as headache, giddiness, insomnia and cold hands.

Digitalis does not raise abnormally the blood pressure in man and if indicated for any reason in those suffering from arterio-sclerosis, it must not be withheld.

#### ARTERIO-SCLEROSIS AND MENTAL DISEASE.<sup>1</sup>

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As a result of the routine examination of patients admitted to the wards of Gladesville and Callan Park Mental Hospitals, it appeared to us more than a year ago that the number showing clinical signs of arterio-sclerosis was unduly high. This seemed worthy of confirmation. We accordingly drew up a schema and agreed to keep a tabulated record of all our new patients who showed considerable deviations from the normal either with regard to changes in the arterial wall or high sphygmanometric readings.

It was felt that independent observations in the two institutions would prove a valuable check on the results. Owing to the fact that we had charge of the admission blocks of our respective hospitals, there would be as material during twelve months, the period under review, the bulk of the certified admissions for the State. There must, however, be excluded from our series all those patients who, being violently maniacal, degraded in habits, profoundly demented or epileptic, were admitted elsewhere.

As the material, therefore, represents the less advanced state of mental disease, it is probable that a total survey of our mental hospital population would yield more striking results.

<sup>1</sup> Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on October 16, 1924.

#### Classification of Arterio-Sclerosis.

We do not propose to discuss here at any length the nature of arterio-sclerosis or the relationship between high pressure and arterial disease, but simply state that we follow the classification which Allbutt has elaborated in his very extensive monograph on the subject.

While insisting that arterio-sclerosis is not a true disease, but an issue common to many diseases, he adopts three main groups. The first is the hyperpictic form in which persistently high blood pressures preceded the arterio-sclerosis and the sclerosis may be limited to the smaller arteries and arterioles. The second group, decrescent arterio-sclerosis, is a heterogeneous class in which arterial degeneration is not typically associated with rise of blood pressure. The third or toxic form is characterized by the effect of certain poisons and toxins as syphilis, typhoid fever, lead, diabetes and so forth. In this group the blood pressure is not necessarily much increased.

According to Allbutt hyperpiesia is typically a process starting between forty-five years and fifty years of age and usually running its course in about ten years. The decrescent form usually starts about a decade later and is compatible with much longer life.<sup>(21)</sup> Allbutt, however, describes many juvenile cases.

In reviewing records a blood pressure of one hundred and forty millimetres in a subject of twenty years has seemed to us as striking as one of one hundred and seventy millimetres in a subject of sixty years and we have regarded the case as one of early hyperpiesia. Blood pressures have been taken by using the auscultatory method with the patient in the recumbent position. We describe the arteries according to the schema shown in Table I., but patients qualifying only for the category I. were not included in the series unless the disease was very widespread or associated with raised blood pressure. We are indebted to Mr. H. W. Apperly, General Manager of the Australian Mutual Provident Society, for putting at our disposal some valuable data. The chief actuary, Mr. Elliott, provided us with the accompanying tables showing normal blood

TABLE I.  
*Authors' Method of Classifying Degrees of Thickening of  
Radial, Brachial and Temporal Arteries.*

|   |    |    |                                       |
|---|----|----|---------------------------------------|
| 0 | .. | .. | Arteries not palpable                 |
| 1 | .. | .. | Arteries definitely palpable          |
| 2 | .. | .. | Arteries markedly thickened           |
| 3 | .. | .. | Arteries hard, tortuous and locomotor |

TABLE II. (A).  
*Average Blood Pressure in the United States and Canada  
in Men.*  
(Millimetres of Mercury.)

| Age.       | Systolic. | Diastolic. |
|------------|-----------|------------|
| 20 .. .. . | 120 ..    | 80         |
| 25 .. .. . | 122 ..    | 81         |
| 30 .. .. . | 123 ..    | 82         |
| 35 .. .. . | 124 ..    | 83         |
| 40 .. .. . | 126 ..    | 84         |
| 45 .. .. . | 128 ..    | 85         |
| 50 .. .. . | 130 ..    | 86         |
| 55 .. .. . | 132 ..    | 87         |
| 60 .. .. . | 135 ..    | 89         |



TABLE II. (B).  
Average Systolic Blood Pressures (Millimetres of Mercury).

| Ages.            | New York Life Insurance Company's Experience. |            | Australian Mutual Provident Society's Experience. | American Standard. |
|------------------|---|------------|---|--------------------|
|                  | South America.                                | Australia. | Australia.  |                    |
| 15 to 27 .. .. . | 118   | 127        | 125   | 120                |
| 28 to 37 .. .. . | 123   | 129        | 131   | 124                |
| 38 to 47 .. .. . | 125   | 130        | 132   | 127                |
| 48 to 57 .. .. . | 130   | 135        | 138   | 132                |

The figures for Australia are based on 250; Australian Mutual Provident, 521; United States and Canada, 250,000 and South America, 500 lives.

TABLE III.  
Mental Hospitals, Callan Park and Gladesville.

| Age.        | Total Admissions. | Hyperpietic Form. | Decrescent Form. | Toxic Form. |
|-------------|-------------------|-------------------|------------------|-------------|
| 0 to 20 ..  | 23                | 3                 | 5                | 0           |
| 21 to 30 .. | 130               | 16                | 19               | 3           |
| 31 to 40 .. | 154               | 16                | 23               | 14          |
| 41 to 50 .. | 140               | 16                | 24               | 16          |
| 51 to 60 .. | 82                | 21                | 15               | 8           |
| 61 and over | 39                | 3                 | 21               | 0           |
| Total ..    | 568               | 75                | 107              | 41          |

pressure and age groups (see Table II.). We are also indebted to Dr. Scot-Skirving for further information.

#### Observations and Illustrative Cases.

Working on these lines then, out of three hundred and ten patients admitted to Callan Park 40%, and out of two hundred and fifty-eight patients admitted to Gladesville 37%, showed sufficiently well-marked signs of arterial disease for inclusion in our series. Records were obtained in practically all patients except a few who were too excited or resistive to permit of accurate sphygmomanometer readings being made. The patients were not in any

TABLE IV..  
Showing Blood Pressures in Hyperpietics (Millimetres of Mercury).

| Age Groups.        | Number in Series. | State of Arteries. |   |   |   | Average Blood Pressure. | Highest Blood Pressure. | Lowest Blood Pressure. |
|--------------------|-------------------|--------------------|---|---|---|-------------------------|-------------------------|------------------------|
|                    |                   | 0                  | 1 | 2 | 3 |                         |                         |                        |
| 20 and under .. .. | 3                 | 1                  | 2 | — | — | 143: 92                 | 150: 85                 | 135: 90                |
| 21 to 30 .. .. .   | 16                | 7                  | 4 | 4 | 1 | 154: 88                 | 180:100                 | 140: 84                |
| 31 to 40 .. .. .   | 16                | 6                  | 7 | 3 | — | 161: 98                 | 210:140                 | 140: 95                |
| 41 to 50 .. .. .   | 16                | 6                  | 6 | 3 | — | 165:109                 | 190:115                 | 145: 95                |
| 51 to 60 .. .. .   | 21                | 10                 | 1 | 6 | 4 | 189:114                 | 260:140                 | 150:105                |
| 61 and over .. ..  | 3                 | 2                  | 0 | 1 | 0 | 195:111                 | 205:105                 | 175:105                |

TABLE V..  
Showing Blood Pressures in Decrescent form (Millimetres of Mercury).

| Age Groups.        | Number in Series. | State of Arteries. |   |    |    | Average Blood Pressure. | Highest Blood Pressure. | Lowest Blood Pressure. |
|--------------------|-------------------|--------------------|---|----|----|-------------------------|-------------------------|------------------------|
|                    |                   | 0                  | 1 | 2  | 3  |                         |                         |                        |
| 20 and under .. .. | 5                 | —                  | 1 | 3  | 1  | 125:66                  | 130: 80                 | 105:60                 |
| 21 to 30 .. .. .   | 19                | —                  | 6 | 13 | 3  | 131:85                  | 150: 90                 | 112:70                 |
| 31 to 40 .. .. .   | 23                | —                  | 5 | 16 | 2  | 134:85                  | 155: 80                 | 110:60                 |
| 41 to 50 .. .. .   | 24                | —                  | 1 | 17 | 6  | 134:78                  | 160:100                 | 107:69                 |
| 51 to 60 .. .. .   | 15                | —                  | 2 | 7  | 6  | 144:89                  | 165: 90                 | 120:75                 |
| 61 and over .. ..  | 21                | —                  | 1 | 7  | 13 | 148:92                  | 215:140                 | 112:78                 |

TABLE VI..  
Showing Blood Pressures in Toxic Group (Millimetres of Mercury).

| Age Groups.        | Number in Series. | State of Arteries. |   |   |    | Average Blood Pressure. | Highest Blood Pressure. | Lowest Blood Pressure. |
|--------------------|-------------------|--------------------|---|---|----|-------------------------|-------------------------|------------------------|
|                    |                   | 0                  | 1 | 2 | 3  |                         |                         |                        |
| 20 and under .. .. | —                 | —                  | — | — | —  | —                       | —                       | —                      |
| 21 to 30 .. .. .   | 3                 | —                  | 1 | 2 | —  | 136: 80                 | 150: 70                 | 120:90                 |
| 31 to 40 .. .. .   | 14                | 1                  | 6 | 3 | 4  | 141: 79                 | 185:100                 | 122:80                 |
| 41 to 50 .. .. .   | 16                | —                  | 6 | 6 | 12 | 154: 96                 | 215:130                 | 107:70                 |
| 51 to 60 .. .. .   | 8                 | —                  | 2 | 2 | 4  | 162:103                 | 230:140                 | 110:75                 |
| 61 and over .. ..  | —                 | —                  | — | — | —  | —                       | —                       | —                      |

way selected except by the machinery of the hospital's administration which excludes certain types of patients from the admission block wards as indicated above. We classified the patients into age-groups and according to the form of arterial disease as shown in Table III. In the toxic group are included only those in whom syphilis or alcohol seemed to be the direct cause of the arterial degeneration. Tables IV, V. and VI. show analyses of the age groups for each of the types of arterial degeneration, hyperpietic, decreascent and toxic respectively.

Referring to his experience as Commissioner in Lunacy, Allbutt says that while arterio-sclerosis abounded in the asylums, yet high arterial pressures and cardiac hypertrophy were neither frequent nor conspicuous, although excessive blood pressure is the antecedent phase of certain types of cases of arterio-sclerosis.<sup>(1)</sup>

Mott states that in necropsies at the London County asylums he has been struck with the frequency of arterial degeneration and with the proportional infrequency of intra-cerebral hæmorrhage, as compared with his experience and statistics obtained at Charing Cross Hospital.<sup>(2)</sup> Dr. S. E. Jones has informed us that he has made *post mortem* examinations in forty-three cases at the Rydalmere Mental Hospital and found areas of atheroma in 93%.

There are many references in the literature to the association of arterio-sclerosis with manic-depressive insanity.<sup>(3) (4) (5)</sup> Thus Albrecht could demonstrate it in eighteen among fifty-four patients.<sup>(6)</sup> This led us to make an attempt to classify our patients on Kraepelinian lines, but we have found great difficulty in compressing them rigidly into pigeon holes and we believe this to be the tendency in modern psychiatry. However, we found many patients whose condition was typically schizophrenic with well marked sclerosis and pressures considerably or not raised. The following is an example of a hyperpietic case:

Case I.—D.G.G., aged twenty-eight years, single, a labourer, was dull and inert and had not been able to work for eleven months prior to admission. He had constantly shifted his residence on account of delusions of persecution by unseen influence; he had auditory and gustatory hallucinations, many paræsthesiæ, ideas of thought-reading and sexual interference. He has been unemployable in the mental hospital and has deteriorated considerably since his admission. His systolic blood pressure was 153 millimetres of mercury and the diastolic 100 millimetres. The brachial and radial arteries are distinctly palpable.

We are also considerably surprised at the number of patients in our series whom we had to label congenital mental deficient. Most of them had super-added psychoses. (This subject will be referred to again in dealing with abiotrophy as a cause.) We give an example of this type:

Case II.—B.A.C., aged twenty-seven years, single, reached the sixth standard at school, but is said to have been dull, though endowed with a wonderful memory. He has never been able to do more than casual labour and has been in many homes for destitute people. He had been sleeping in the domain for some months prior to his admission and sought refuge at the Board of Health. His mental age was nine years, he was docile and industrious at simple tasks in the hospital, though introspective and complaining of many imaginary illnesses. His systolic

blood pressure was 160 millimetres of mercury and his diastolic pressure 80. His arteries were distinctly palpable.

Sir Maurice Craig and others have found high blood pressures in melancholia and low blood pressures in mania. This generalization is certainly not universally applicable. Some of our typical manic patients had definitely high pressures, though in many of them we failed to obtain reliable readings. Many melancholics fell at the lower end of the end of the scale. We quote two cases:

Case III.—S.J., female, aged fifty-eight years, has cardiac hypertrophy and systolic and diastolic blood pressures of 200 and 110 millimetres of mercury respectively. She had hard arteries and no albuminuria. She has been having recurrent attacks of typical mania with flight of ideas, euphoria and excessive psycho-motor activity and without confusion for the last thirty years. As far as can be ascertained she has had no phases of depression.

Case IV.—P.H., a male, aged forty-two years, married; is a grazier. His mother, one brother and one sister (out of five brothers and three sisters) suffered from insanity. He was transferred from another institution where he had been treated for three months and had required tube feeding. He was said to have made several attempts on his life. He showed definite emotional depression, poverty of ideation and psycho-motor inhibition and had hypochondriacal delusions with regard to his bowels and so forth. He was able to give a coherent account of his past life. The history showed that his illness had been ushered in by a manic phase. The systolic blood pressure was 108 millimetres of mercury and the diastolic 78. The radial, brachial and temporal arteries were all much thickened and tortuous, there was a trace of albumin in his urine and there were minor signs of hypothyroidism.

A small, well-defined group of eight patients all under thirty years of age suffering from considerable mental confusion could be split off. In all of these there was a high blood pressure ranging from 140:100 to 180:100 associated with considerable thickening of the arteries. In seven, bacterial intoxication could be demonstrated. They all recovered.

We have hitherto not broached the subject of classical arterio-sclerotic insanity, as patients suffering from this condition rarely find their way into the admission wards. But in them it is always questionable as to what extent we are dealing with a terminal state and to what extent we are dealing with a disease *per se*. Many of our patients may in time qualify for this classification and if their lives are not terminated prematurely, may provide the striking specimens of cerebral destruction to be seen in pathological museums. On the other hand, it has frequently been suggested that the stresses thrown on the arteries in manic depressive insanity are the cause of their degeneration. It is the old question of *post hoc* or *propter hoc*. In the one case arterio-sclerosis is assigned to the cause of mental disease and in the other *vice versa*. Kraepelin offers a useful analogy, when he says that in manic depressive insanity it may be:

That the same poison which engenders the alteration of psychic states, affects also the arterial walls, just as one thinks of the relation of syphilis, *id est* paralytic, vascular change and the corresponding cortical diseases.

A quotation from Diefendorf also seems apt here:

In arterio-sclerosis insanity the vascular change is an accompaniment of only secondary importance in a disease process which is highly destructive of nerve tissue.<sup>(7)</sup>

We are indebted to Dr. G. B. Wooster for notes of two patients suffering from congenital mental deficiency with associated arterio-sclerosis in the wards of Callan Park.

The two patients are brothers, one twenty-one years and the other seventeen years of age. The family history reveals an intemperate and promiscuous father who refuses to live with his family. The elder brother is dead (tuberculosis) and was mentally affected (probably congenital mental deficiency). One sister is slow mentally and unable to earn her living, one boy and three girls are apparently normal. A paternal uncle is eccentric and probably suffers from congenital mental deficiency. A maternal uncle is an alcoholic and is in a mental hospital.

J.F., aged twenty-one years, the elder patient, is fifth in the family. Labour was difficult and prolonged to five days and without medical assistance. Patient was not backward in physical activities, but did not talk till seven years. He could not be taught anything at school, but used to do some manual work. At home he became subject to attacks of bad temper and he threatened his sisters. The mother was afraid he might subject them to indecent assault.

On physical examination he is seen to be of good physique. The apex beat is in the sixth intercostal space and in the mid-clavicular line. There is no accentuation of the second sound at the aortic area. The systolic blood pressure is 145 millimetres of mercury and the diastolic pressure 100. The brachial, radial and temporal arteries are all distinctly thickened. His serum has not reacted to the Wassermann test. He manifests the following stigmata of degeneration: His palate is high and arched, with a deep longitudinal furrow, but there is no definite cleft. The ears are large and prominent, the face asymmetrical and the hands large and broader than normal. He is obviously weak-minded and incapable. His mental age is seven years and two months. He grins in a senseless fashion, his speech is defective and he cannot read or write. He does some work about the hospital and is usually amenable to discipline.

R.F., aged seventeen years, the younger patient, suffers from still more obvious mental defect. He is underdeveloped and has some degree of scoliosis. The apex beat is in the fifth intercostal space, 1.25 centimetres outside the mid-clavicular line. The heart sounds are clear and forcible and there is a diffusive heaving pulsation of the whole precordium. The aortic second sound is accentuated. The pulse rate is 95. The systolic blood pressure is 136 millimetres of mercury and the diastolic pressure 100. The arteries are very thickened. The brachial artery is tortuous. His extremities are blue and cold. His serum has not reacted to the Wassermann test. There are definite nystagmoid movements of the eyes. Stigmata of degeneration are present. He has defective dentition and also narrow and arched palate, mouth breathing, prominent ears with well marked tubercles, asymmetrical face, short fifth finger, large fontanelles. Mentally he is very dull, simple and childish. He is very easily managed. He could not accept any education at all, but can do simple tasks. His attention is wandering and his aspect dreamy and stupid. Binet Simon tests give intelligence of 7 years. His speech is very defective, he will not carry on a conversation and talks in monosyllables.

We have quoted these two cases more fully as they are remarkably clear examples both of arterio-sclerotic changes and of mental deficiency. The fact that these phenomena occur in brothers suggests that the germ plasm is at fault.

We look upon the association of mental disease with arterio-sclerosis in general as one not of simple cause and effect, but common results of many detrimental factors operating on the organism. Let us consider what these factors may be.

#### Factors Concerned in the Relationship Between Arterio-Sclerosis and Mental Disease.

##### *Abiotrophy.*

All writers on mental and arterial disease insist upon the importance of heredity in their respective

domains. The frequency of arterial disease in young subjects of congenital mental deficiency and of schizo-phrenia in our series suggests that deficiency of the "vital rubber" and deficiency in the neurones may both be expressions of an effete germ plasm to which we apply the term abiotrophy. It might be useful in this connexion to discuss the process of involution in general. Some of our patients become grey and wrinkled, some show *arcus senilis* and look much older than their years, suggesting a premature senility.

##### *Mechanical Causes.*

Heavy muscular work is often cited as a cause of arterio-sclerosis. It would not, however, appear to be a very potent one among our patients, though it may be a factor in a few. Certainly many have done very little manual work. We have already dealt with the restless activity of the maniac considered a factor by some authorities.

##### *Psychic and Mental Causes.*

Psychic strains and mental conflicts, all of which may be considered under the convenient expression "worry," doubtless play their part. The subject of anxiety and suprarenal over-activity with its pressor effects on the circulation is still *sub judice*, though it has received some experimental confirmation.

##### *Auto-Intoxication.*

In regard to auto-intoxication we have first to consider the question of endogenous toxins produced by faulty metabolism of the tissues themselves. Batty Shaw found in the kidneys substances which had pressor effects on the arteries. To these he gave the name "rennin."<sup>(8)</sup> Stoddart suggests hypothetical auto-toxins as the causes of mania and melancholia.<sup>(9)</sup>

##### *Micro-Organisms.*

More important is the question of toxic bodies produced by micro-organisms, especially those inhabiting the intestinal tract. We have endeavoured to collect evidence on this point. B. H. Shaw, of Stafford Asylum, has cultured organisms from his patients and shown that these organisms produce appreciable amounts of formaldehyde.<sup>(10)</sup> In experiments on rabbits Loeb found that aldehydes of the aliphatic class only were followed by arterio-sclerosis with a high degree of certainty.<sup>(11)</sup> E. Mellanby has discredited the importance of the indoxyl group in arterio-sclerosis. On the other hand, we found an indican reaction in the urine of 31% of our patients, while a similar reaction was found in only one out of sixty members of the staff at Callan Park.

##### *Hypo-Chlorhydria.*

As hypo-chlorhydria has been cited as a factor in increasing bacterial growth,<sup>(12)</sup> a small series of fractional test meals amounting to eighteen in number was undertaken from arterio-sclerotic subjects at Callan Park, specimens being drawn off at quarter hour intervals for three hours. In five patients the combined acidity was above normal, in the rest it was distinctly below. Free hydrochloric acid was absent in two instances, the level lay between an average of 2.16 and 13.6 (normal 16) in eleven; this is



low. In one it was just above the average and the remainder showed a high amount of free hydrochloric acid. These results are shown in the accompanying chart.

#### Oral Sepsis.

Oral sepsis is extremely common in mental hospitals, but it was not possible to draw any conclusions as to its relation to the special class of cases under consideration.

#### Exogenous Poisons.

Exogenous poisons, such as lead, arsenic, alcohol must next be considered. We have one doubtful case of plumbism and possibly one case of arsenical poisoning in our series. Alcohol, however, has lived up to its reputation; no less than 80% of habitual drinkers have well-marked arterio-sclerosis. On the other hand, some of the patients suffering from the more grave forms of alcoholic dementia have arteries that cannot be palpated with blood pressures well within normal limits. This observation is suggestive as showing that we must look for more than one aetiological factor in any given case.

#### Infections.

Andrewes has demonstrated diffuse and focal lesions in the vessels in many infectious fevers, such as typhoid and rheumatic fever and also pyogenic infections.<sup>(13)</sup> It is naturally difficult to evaluate these factors among our patients, but several give suggestive histories, the chief offenders being those suffering from pneumonia (four cases), influenza (three cases), appendicitis (four cases).

As regards septic infection the following seems a striking case:

Case VII.—H.W.J., a male, aged twenty-seven years, was a voluntary patient. He was admitted for psychasthenic symptoms; he had a blood pressure 150:80, hard and thickened arteries and had been suffering for some time from multiple furuncles. He did well on autogenous vaccine.

#### Syphilis.

Although atheroma can be almost universally demonstrated on the *post mortem* table in general paralytics, in our patients, generally not in advanced stages, arterial disease was somewhat less frequently demonstrable than among the total of newly admitted patients. When present the disease was of the decrescent form except in those patients whose condition was complicated by alcoholism; some of these had excessive blood pressures. The low systolic and diastolic readings were striking in many; the lowest reading at Callan Park was

93:45 and at Gladesville 98:62. We show table obtained from the Gladesville patients (Table VII.). In cerebral syphilitics, however, as might be expected clinical arterio-sclerosis of the decrescent form is much commoner.

#### Excessive Protein Diets.

We are unable to offer any useful observations on the subject of excessive protein diet, except that we have restricted proteins and salt in a number of instances without striking results.

#### Ductless Glands.

Allbutt says that senile arterio-sclerosis, especially when of the precocious form, may be due to thyroid deficiency.<sup>(14)</sup> McCarrison states that arterio-sclerosis is a common feature of myxedema.<sup>(15)</sup> Fishberg quotes a patient with generalized arterio-sclerosis who suffered from thyroid deficiency.<sup>(20)</sup> Taking Hertoghe's eyebrow sign, coarseness of the skin, malar flush, slow pulse and so forth as signs of minor hypothyroidism, fifty-one patients of the Callan Park series were placed in this category; and this view was corroborated by their tolerance of thyroid feeding. A *post mortem* examination in one instance revealed considerable fibrosis of the thyroid. We have formed no conclusions on the controversial subject of the overaction of the suprarenal and pituitary glands.

#### Blood Changes.

Blood counts were done in a number of instances at Callan Park; forty-two red cell counts were made, fifty-one white cell and forty-nine differential counts. Out of the forty-two red cell examinations eighteen yielded

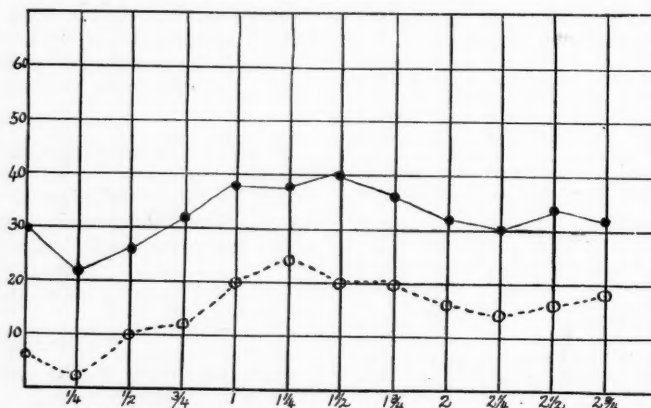
TABLE VII.

Showing Blood Pressure in General Paralytics (Gladesville Series).<sup>1</sup>  
(Millimetres of Mercury.)

| Name.       | Age. | Blood Pressure. | Arteries. | Alcoholism. |
|-------------|------|-----------------|-----------|-------------|
| E.M.J. ...  | 36   | 119: 79         | 0         | —           |
| C.C.S. .... | 37   | 120: 83         | 0         | +           |
| S.G.W. ...  | 38   | 117: 79         | 1         | —           |
| H.W. ....   | 43   | 107: 70         | 2         | —           |
| G.A. ....   | 45   | 215: 130        | 2         | ++          |
| W.R.S. ...  | 45   | 98: 62          | 0         | —           |
| E.E.W. ...  | 45   | 124: 84         | 1         | —           |
| C.J. ....   | 46   | 155: 95         | 2         | —           |
| D.M.A. ...  | 52   | 162: 108        | 0         | —           |
| C.G. ....   | 57   | 130: 80         | 1         | —           |

<sup>1</sup> This table includes all cases irrespective of arterio-sclerotic changes.

CHART SHOWING THE RESULT OF EIGHTEEN FRACTIONAL TEST MEALS ON ARTERIO-SCLEROTIC SUBJECTS.



Ordinate = Time in hours.

Abscissa = Acidity of test meal in terms of deci-normal hydrochloric acid.

The continuous line denotes combined acidity.

The dotted line denotes free hydrochloric acid.

results between 4,500,000 and 5,500,000 cells to the cubic millimetre. Six were above this limit and eighteen below. Out of fifty-one white cell counts fifteen yielded results between 7,000 and 9,000 per cubic millimetre. Sixteen were above this and twenty below. The average count in forty-nine differential estimations was as follows: Polymorphonuclear cells, 61%; large lymphocytes, 12%; small lymphocytes, 23%; eosinophile cells, 3%; basophile cells, 1%.

These figures show a relative lymphocytosis and some anaemia. Kraepelin quotes authorities who report somewhat similar findings in manic depressive insanity and *dementia præcox*.<sup>(17)</sup>

#### Relation to Bright's Disease.

Dr. Latham estimated total nitrogen, urea and creatinin in four cases of the juvenile type and found the percentages to be within normal limits.<sup>(18)</sup> However, further observations of this type would be instructive. A trace of albumin was present in the urine in 33% of our patients and a few casts, usually hyalin or granular, were present in 15%. This did not seem to us sufficient evidence of renal disease in most instances. This view is confirmed by Mott's observations, that in necropsies on one hundred and sixty asylum cases of arterio-sclerosis some degree of interstitial fibrosis of the kidneys (usually not very extensive) was found in only sixty-five instances.<sup>(19)</sup> We therefore follow the opinion of many authorities, that Bright's disease and arterio-sclerosis are not necessarily associated. Five of our patients had definite symptoms of Bright's disease, but we find it difficult to attribute their mental symptoms entirely to the failure of kidney functions, as many people with advanced Bright's disease manifest no psychosis. It seems feasible that the noxe which damaged the kidneys, simultaneously attacked the brain and the arteries.

#### Conclusions.

What conclusions are to be drawn from the foregoing? We have presented the above ætiological picture merely as a schema of lines along which we think further investigations might be conducted. It is a subject in regard to which we must confess to feel very much in the dark. What we hope to have helped to establish is that arterial disease is extremely common in mental hospitals, even at very early ages and that it is not the prerogative of any particular psychosis. At the same time we have no doubt that many mental patients of the same psychotic type go through life with very healthy arteries and that many subjects of severe arterial disease still retain the highest intellectual capacities. It seems then that arterio-sclerosis and mental disease are rarely related as cause and effect, but rather are common effects of a heterogeneous group of causal agents operating in predisposed subjects. The causal agents are of the type that attacks the organism as a whole, rather than of those that exercise a specific action on any one tissue or group of tissues. In such an attack it is not surprising that the controlling cells of the body (the nerve cells) and the irrigating channels should show signs of injury. Sometimes it may be

a pure case of degeneracy; "the *élan vital*" is so weak that the organism withers, so to speak, or at least is by no means fitted to stand stresses which a sturdier one would pass through unscathed. It may be that the germ plasma was originally of poor quality or that it has been damaged before conception by such influences as alcohol, syphilis, tuberculosis or other toxins in the parents. The developing organism may again be damaged *in utero* or after uterine life, when it has to meet stresses in the shape of infections, toxins and the subsequent interaction between the various tissues. Or again when the organism begins to function as an individual, it fails to adapt itself to many conditions in its environment on account either of the very unfavourable nature of this environment or of its own poor capacity of adaptation, that is, causes of the order which we call psychological.

#### Prognosis and Therapeutics.

As to the prognostic and therapeutic indications of these associated conditions, we are able to say nothing at present. Data of this kind can only be obtained by systematic observation over a number of years.

#### Acknowledgements.

We are indebted to Dr. Eric Sinclair, Inspector-General of Mental Hospitals, Dr. H. C. MacDouall and Dr. J. A. L. Wallace for permission to present this paper. We would like also to thank the nursing staff for their ready cooperation.

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## HYSTERICAL DELIRIUM.

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ROSANOFF<sup>(1)</sup> in his "Manual of Psychiatry" describes hysterical states as "episodic mental attacks in the forms of delirium, stupor or dream states during which repressed wishes, mental conflicts or emotional experiences detached from ordinary consciousness break through and dominate the mind." In this paper I intend to confine my attention to hysterical delirium—six cases of which have come under my notice during the past six months. Hysterical delirium, occurring as it does in various forms, somnambulisms, fugues, dual personalities and the like, has been extensively studied during the past thirty years. It is considered by Jung<sup>(2)</sup> and Janet<sup>(3)</sup> to be fairly common and its study has thrown much light on the mechanism of hysterical phenomena.

The first patient I wish to bring under your notice, is a young married woman in good physical health. She has one child. Her life she considered uneventful but happy, until about six months before admission, when she began to be worried by the not unwelcome attentions of a new lover. A strong conflict was thus aroused within her. For she was of a religious nature, she had a keen sense of her marriage obligations and of her duties to her husband. On the one hand, she felt inclined to follow the dictates of her heart; on the other hand, her religion, her marriage vows, her sense of duty prevented it. She became ill; her illness was functional in origin. It was characterized by generalized weakness, fits of screaming, *astasia-abasia* and the like. All through this illness she was tormented by the imperative thought that her husband must be told all. Pride and shame prevented it. One night, however, when her husband was beside her bedside she told him everything. The telling was dramatic—she acted all, she emphasized all and yet the whole delirium was against her own will. "I couldn't help myself," she says. As a spectator in a theatre, she sees her own wishes and desires dramatized before her eyes and she is powerless to stop the play. She retains a vivid recollection of the whole incident.

In this patient there existed for some time on the "threshold of consciousness," the powerful system of ideas that her husband should be told all. This thought ever sought for adequate expression, but was always suppressed. Yet eventually in spite of herself, it dominated her consciousness and was given expression to.

My next patient is also a young married woman whose married life has been far from happy. Her husband is a drunkard and has often insulted, abused and even assaulted her. A fortnight after her marriage she developed a number of phobias. In fear and trembling one day she hid a razor in a cupboard fearing that she might cut her throat. She lived then in a state of chronic fear. Always she feared some impending harm. She thought that she might cut her throat, her husband's throat, her baby's throat. In spite of repeated holidays in the country, her fears became steadily worse. Eventually many months before admission she became ill and was confined to bed. She had a general sense of weakness; she was unable to walk without assistance, although there was no apparent weakness of the legs (*astasia-abasia*). And as was demonstrated on admission, with her eyes shut she could walk perfectly well. At times she had attacks of scream-

ing and often she told her people about the quarrels she had had with her husband. On admission she was physically strong. She had exaggerated knee jerks. There was noted besides a transient erythema of the neck, which at times could be provoked by suggestion. She had definite dermatographia, the wheals persisting sometimes as long as two to three hours. During her stay in hospital she had a number of deliria, during which she dramatized practically the whole of her life. These deliria could be aroused at any time by suitable suggestion. By a suitable stimulus word the patient would become an actress of the highest order. In words wonderfully arranged and wonderfully expressed, with actions vividly expressive of the intense emotions swaying her, she would dramatize incidents of her past life. The following delirium aroused by the stimulus word "baby" represents the climax of a quarrel she had had with her husband:

He came in one day talking fight—his neck was covered with blood (folds her kimono around her neck). I mustn't run away or do anything wrong (picks up a picture of a little nigger and kisses it)—Oh, my baby—Oh, my baby. He had a knife in his hand (she jumps up violently, puts her leg on a seat, takes off her shoe, rushes over to the door in a crouched attitude, as if protecting her baby. She cries bitterly) Oh, my baby—Oh, my baby.

The whole personality of the patient is in abeyance during this scene. Her whole attention is devoted to her exquisite acting. She suffers from visual illusions, which Jung<sup>(2)</sup> insists are characteristic of this condition; she mistakes the photograph of the nigger for her baby. She is accessible, however, during the delirium, at any time it can be terminated by command, at any time it can be re-aroused.

This patient is a woman who has struggled for years against a most unfavourable environment. During this time a number of most painful scenes were impressed and re-impressed upon her mind. They made a most powerful impression on her; the memory of them is vivid. At any time they can by suggestion be so brought to the surface that they dominate her whole personality and are wonderfully acted again in all their old vividness by a patient who has no pretence of being an actress.

My third patient is a young unmarried woman from whom I find it impossible to elicit much useful information. At times she has peculiar hysterical fits. When asked to think of "mother," she becomes rigid, her attitude is one of paralytic fear, she gives evidence of visual hallucinations, she stares up in the air and says plaintively: "Mother, mother" (her fear intensifies) "don't make me drink that," she implores. She clutches at her abdomen. She then lies on her back exhausted. The fit lasts all told two to three minutes. It is repeated in the same way time and time again.

In this patient a grave emotional experience of the past lies stored up in her mind in such a vivid form that given a suitable stimulus it springs into activity once again and assumes absolute control of her personality and she is unable to prevent the process.

The next patient displays a much more profound disturbance of personality than the other three. She is a young unmarried girl, her life has been quiet and secluded, she neither sought company nor amusement. Some time before admission she experienced a great psychic shock; it was an assault. After a series of vicissitudes she developed amnesia both retrograde and anterograde. She regressed to an infantile state. She became an infant or four again. She tenderly nursed a doll. She was robust and well developed and had no organic illness. She had complete anaesthesia of the whole body; even the cornea

<sup>1</sup> Read at a meeting of the Section of Neurology and Psychiatry of the New South Wales Branch of the British Medical Association on October 16, 1924.



were anaesthetic. The scalp and pubic areas, however, were hyperaesthetic. There was complete amnesia for her whole life. She did not remember even going to school. She showed the interesting phenomenon of automatic writing. At irregular periods during her stay in hospital her assumed personality became forgotten and she lived once again scenes of her past life. Now she was a typist and wrote business letters on an imaginary typewriter, now she talked and gossiped with a girl friend, but more especially were prominent the scenes connected with her psychic shock. After this incident she had determined to throw herself over the Gap, but was restrained on the brink by a man who made her promise never to do it again. The delirium commenced one night and was continued as follows at exactly the point left off on the following night.

She gets out of bed. "What's the use of living, there's not much in life." Opens the door, closes it, walks out on to the verandah, then back into the room. "Better not catch a tram, I may be seen." Now she stands on a chair, steps on to the wash stand to throw herself over the Gap. Talks to the man who interferes. "It is nothing to do with you. Think it is like your cheek. It takes me all my time to mind my own business. It's nothing to do with you. I can please myself what I do." Laughs. "Fancy you, a black man, to speak of this as a white world. It seems so funny. Thought I was mad. Oh, but the water calls to me. Oh, well, if it comes to that I'll promise not to jump in, but to go back." Cried for ten minutes.

The delirium lasted two to three hours. On its completion she went into a profound sleep and on awakening had no knowledge whatsoever of what she had acted during the night. During the delirium she was totally inaccessible, she could not be aroused even by pin-pricks; she was totally unaware of her environment. She is a dual personality akin to Janet's famous *Irène*.<sup>(4)</sup> Her personality in hospital was an assumed infantile one, yet her own personality, sunk in her unconscious mind, is ever ready to break through the barriers that retain it and does so in delirium and in scenes such as I have quoted above. The past having dominated her personality for a time, is once more thrown back into her subconscious mind and the patient forgets the delirium she has just acted.

These four women are very similar to one another. For years their lives were smooth and uneventful and whilst so they remained well. When difficulties and emotional shocks were encountered they became ill. Even though well nourished and physically normal, they were incapacitated for months by an illness that was in reality an advantage to them.

In all four powerful systems of ideas were operating and ever seeking expression. In three of them the systems were suppressed, they knew how foolish it would be for them to broadcast, as it were, the ideas dominating them. In the fourth patient there was complete suppression into the subconscious mind of all her past life and doings. Yet in spite of themselves, nay, in two of them at the will of the observer, they saw their own thoughts and desires dramatized before their eyes and they were powerless to prevent it. How then can these deliria be explained? We know that in toxic conditions, as acute infective processes, alcohol, plumbism, *encephalitis lethargica*, we get delirium of various kinds. The accepted modern interpretation of such deliria is that the toxins damage the cells of the frontal and prefrontal areas, leading in some cases to permanent chromatolysis of the cells and permanent mental disease.<sup>(5)</sup> Once these cells are injured by toxins their inhibitory power is damaged, the lower centres assume control and deliria and other psychotic manifestations appear.<sup>(6)</sup> But there

can be no question of a toxæmia in these patients. It is true that they were ill and that they were confined to bed, but their illness had no physical basis. It was simply a subconscious refuge from the difficulties that confronted them in life. Surely refuge of this kind must be regarded as most abnormal. Instead of facing their difficulties squarely they enlist the aid and sympathy of others by an illness which is very real to them.

Emotional shocks of great severity it is true will often induce signs of hysteria in even normal men and women, but in these patients hysterical stigmata could be aroused with the greatest of ease. They have thus a predisposition to hysteria, they belong to the "psychopathic personality" group of modern psychiatrists. In them there thus appears to be an inherent and as yet unexplained weakness in the highest cells of the frontal and pre-frontal areas that constitute personality. They may have the normal amount of intelligence, their thinking powers, their scholastic attainments, all signs of great cerebral development may be normal and yet at times from trivial psychological causes there is a reversion to a pure thalamic type of conduct (uncontrollable laughing, crying, fears, mania and the like). Bleuler even suggests that the exaggeration of the tendon reflexes in hysteria is a sign of this weakness of the higher cortical cells.<sup>(7)</sup>

In toxæmias when these cells are injured we get deliria of various kinds, so from the labile character of the cells in hysteria we get various hysterical phenomena. Now these phenomena vary from patient to patient and depend on the contents of the subconscious mind whose existence and importance was first emphasized by Freud. The vast literature on the subject is full of cases proving that various hysterical paralyses, fugues and their like were the direct results of past emotional experiences, repressed wishes and desires and psychic traumata. So also in these four patients the contents of their deliria were the direct result of wishes and desires and emotional experiences of the past. But we must admit that these desires and experiences would not be sufficient to cause deliria in normal people. So once again we fall back for an explanation to the question of psychopathic personality. On this matter we see a vast literature of psychological supposition, abstract thinking, the influence of the higher cortical cells on personality, the influence of mind over matter and the answer we get, is purely an agnostic one: "We do not know."

If we examine these patients, however, we see that intimate details of their lives were told and they were powerless to prevent it. It appears thus that their power of volition was deeply affected. They no longer had control over their actions. But nobody has ever been able to tell us how we control our actions or what we mean by "volition."

Charcot suggests the loss of control is due to abnormal suggestibility; Janet and his followers emphasize the dissociation and emancipation of ideas that constitute personality. Freud says that it is due to sexual elements of various kinds. Whilst these theories afford us a working basis to explain the mechanism and production of hysterical pheno-

mena they do not explain the ultimate cause of hysteria.

On the minds of these four patients were impressed vivid scenes of the past. They tried to control these scenes—to suppress them—their self-control was lacking and the scenes at times would dominate their field of consciousness and be acted in all their vividness in the form of deliria. It is a reversion to an atavistic kind of conduct, when the thoughts of men were more expressed by symbols and actions rather than speech.<sup>(1)</sup>

To sum up, hysterical deliria teach us much about the production of hysterical phenomena. They occur practically solely in psychopathic patients. They are a dramatization of past emotional experiences, repressed wishes and desires and psychic traumata.

#### Acknowledgements.

I wish to acknowledge my indebtedness to Dr. Evan Jones, Superintendent of Broughton Hall, for his advice in the preparation of these notes.

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### Reports of Cases.

#### CLINICAL NOTES ON A CASE OF POLYCYSTIC DISEASE OF THE KIDNEYS AND LIVER.

By H. I. HOLMES, M.D., Ch.B. (Melbourne),  
Warrnambool, Victoria.

CASES of polycystic disease of the kidneys and liver are comparatively rare. This patient having been under observation for a period of seven and a half years, I deemed a few notes and observations made during that period to be of interest. In THE MEDICAL JOURNAL OF AUSTRALIA of January 19, 1924, Professor Cleland, of Adelaide, is recorded as having described at a meeting of the South Australian Branch a series of five cases recently found *post mortem*. I would refer readers of these notes to this publication and also to Cabot's "Modern Urology" for a further description of the disease.

#### Clinical History.

I first attended Mrs. B., on October 22, 1907, in her first confinement, when with forceps she was delivered of a female child; recovery was normal, except for a slight rise of temperature for three days.

I did not see her again till November 4, 1916, when, her medical attendant having failed to attend, I was sent for. She was then forty years of age, was four months pregnant and had an irregular fever (which her medical attendant had remarked as resembling malaria in type). She also had pain in the right side which I entered as "kidney or colon." I visited her only once.

My next visit was on March 9, 1917; she was then eight months pregnant, urine was scanty and loaded with albumin. She was transferred to hospital. On admission the temperature was normal, the pulse rate 90 and the

respiratory rate 40 to the minute. She was distressed at times. Under treatment the improvement was rapid, the urine increasing from three hundred cubic centimetres (fourteen ounces) to two litres (seventy ounces) *per diem* and the albumin rapidly diminishing. She was discharged from hospital after three weeks. One week later she had normal delivery of a female child (her third), the second child having been still born at eight months. Recovery was normal and she nursed the baby for six months.

Three months later on July 27, 1917, I was called to see her again. There was still a good deal of albuminuria, also a note entry of "pus" and a tumour in the left renal region. I visited her on three occasions between this and February 3, 1918, when the renal tumour was still present, also an enlargement of the liver in the epigastrium. Suspecting hydatid, I had an X-ray examination made. A fortnight later she was admitted to hospital and an operation was performed. Both kidneys were enlarged, the left more so. A fairly large cyst, about the size of a small mandarine orange was removed from the left kidney by the trans-peritoneal method and a smaller one from the right lobe of the liver in the epigastrium. The fluid did not yield a reaction when tested for hydatid and a subsequent diagnosis of polycystic disease of the kidneys and liver was made by Professor Allen.

While in hospital the temperature was irregular; high at times and followed by sweats. No clinical records were made at this time or after several subsequent admissions, as it was war period and the resident medical officer was on active service and everybody was working under pressure.

Three months later she was complaining of pain in the liver wound and soon after had an attack of "influenza."

Owing to financial considerations visits were only made when sent for, but in October, 1918, March, May and June, 1919, she was attended for what the patient described as "liver attacks," accompanied by dyspnoea and fever running to 38.9° to 39.4° C. (102° to 103° F.), only lasting a few days and apparently responding to quinine and salicylates.

On January 20, 1920, she was three weeks over menstrual time; the specific gravity of the urine was 1014; no albumin was present. Intermittent feverish attacks occurred during the ensuing twenty months till I saw her again on August 6, 1921, for a more severe attack.

On December 19, 1921, another severe attack required attendance. The temperature was 40° C. (104° F.) and the pulse rate 120 in the minute. She was treated with salicylates and the condition subsided in four days.

Another severe attack, March to April, 1922, was followed by a period of nine weeks in hospital, during which two hundred and fifty cubic centimetres (eight ounces) of fluid were on one occasion drawn off by needle in the right lower axillary area. At the end of this period she left hospital at her own request. The temperature was still irregular and above normal.

After this she did remarkably well, gained weight and looked much improved till she got "influenza" followed by pneumonia at the right base, for which she was in hospital three weeks. Again she left too soon and returned in a few days with pain in the right side.

During these periods there had been only a trace or no albumin in the urine, the kidneys had not increased, but rather diminished in size; the liver had varied; cystic change had apparently been occurring with subsequent shrinking.

On April 12, 1923, she had still been having feverish attacks, had been jaundiced at one period and slightly on another occasion. An X-ray examination revealed the liver much enlarged with a darker shadow below the right dome; the heart was small. No albumin was found in the urine. Four days later the fever was still persisting, there were cramp-like pains "all through the liver," nausea and on one day vomiting, undigested food and no bile in the stools, so I insisted on her return to hospital. With a needle a small amount (fifty to one hundred cubic centimetres) of clear limpid fluid was aspirated from the liver, but no large amount could be located. The fluid was alkaline, slightly albuminous and did not react to the complement deviation test for hydatid. She again left hospital with a slight rise of temperature.

On May 25, 1923, the temperature was 37.9° C. (100.2° F.) and the pulse rate was 108 in the minute; she was perspiring freely at night and losing weight. Her menses were scanty and irregular.

On June 3, 1923, on re-admission to hospital the liver was four finger-breadths below the costal margin and the upper dullness was in the fourth right interspace in the nipple line. The enlargement was not tender. There was free fluid in the abdominal cavity. Diarrhoea with painful defecation had occurred several days previously.

For notes from July, 1923, till death I am indebted to Dr. Godbehear.

In July, 1923, the patient was emaciated, with a dry, pasty complexion and a conjunctival tinge of jaundice. The liver was enlarged with irregular nodules below the costal margin. The kidneys were enlarged and palpable. The spleen was not palpable. The abdomen was distended. No oedema was present in the lower limbs. Urine for twenty-four hours amounted to 1,200 cubic centimetres. Its specific gravity was 1020, it was acid, contained no albumin, but some bile. Microscopical examination revealed scanty pus cells, leucocytes, epithelial cells and granular casts; but a later examination revealed no pathological cells. Faeces were clay coloured and constipated. Blood examination was made when the temperature was highest in the evening. The leucocytes numbered 12,500 per cubic millimetre and the erythrocytes four and a half millions; no parasites were seen and neither lymphocytosis nor leucocytosis was present.

Between July 27, 1923, and February 23, 1924, the abdomen was tapped sixteen times and fifteen litres of fluid were drawn off. The fluid contained 1% albumin (Esbach), it contained bile and did not react to the Wassermann test. The patient used to return home between the tapplings till too ill to do so. The amount of urine daily was 1,200 to 1,500 cubic centimetres, with an occasional trace of albumin. There were intermittent attacks of diarrhoea accompanied by abdominal pain. The veins of the abdominal wall greatly engorged. The patient passed through a period of mental irritability into unconsciousness and died practically from exhaustion on February 23, 1924, or six years from the time the condition was diagnosed.

#### Pathological Report.

*Post mortem* examination revealed a soft web adhesion of the right lung to the chest wall; no induration or thickening of lung tissue was present. The heart was small and no definite pathological change was found in it. The liver was much enlarged and the right lobe more changed than the left; the left lobe was felt to contain a fairly large cyst and several smaller cysts on its under surface; the right lobe was almost entirely involved in the cystic change and closely adherent to the right kidney which was removed with it. The gall bladder was distended with bile; no gall stones were present. On superficial examination each kidney appeared to be almost entirely involved in the cystic change, the surface being studded with cysts from the size of a pea to a fair-sized mandarin orange and looking something like bunches of grapes embedded in a web. The bladder appeared normal. Indurated areas were felt in the spleen, but no cystic change was seen. The uterus and appendages were normal. Large and small intestine contained

no obvious pathological changes. A few enlarged lymph glands were present along the spine, on the diaphragm and in the mediastinum. A small mass of tissue was found in the left inguinal canal; this had been tender during life and present for years.

For the microscopical report I am indebted to Dr. Bull, of the Bacteriological Laboratory at the University of Melbourne. The surface of the liver was irregular owing to the presence of many cysts varying in size, but averaging 3.7 centimetres in diameter. The cysts were irregularly distributed and in portions of liver free from cysts the surface was smooth, the capsule was not thickened, but there is a uniform increase in fibrous tissue rather of unilobular type. The cysts were full of clear mucinous material without cells and with no signs of lining epithelium. There was a good deal of fibrosis around the cysts. Everywhere the bile ducts showed signs of proliferation and multiplication and all stages of cyst formation from tiny dilatations up to large cysts were present. The cysts appeared to have originated in the hepatic ducts. The hepatic cells showed slight fatty change.

The cortex of the kidneys contained large numbers of irregular cysts consisting of mucinous material, but no cells. Kidney tissues manifested fine fibrosis of old inactive type, although there were a few tiny patches of round cells.

Malpighian tufts were inactive and did not fill Bowman's capsules. The tubular epithelium was largely degenerate and cysts seemed to have originated in the tubules.

The spleen was slightly enlarged and was the seat of a fine fibrosis with thickened capsule. There were two well-marked infarcts present which had not undergone fibrosis.

A small piece of tissue showed old fibrosis with cysts and there was one nodule of lymphoid tissue showing fibrosis.

The kidneys did not appear to have much healthy functioning tissue.

#### Comments.

Very little seems to be known of the nature or aetiology of this

condition; in some medical works only passing reference is made to it. Observation of this case, however, certainly makes me conclude that the causative factor may be a filtrable or unrecognized virus and that the condition is not of congenital origin to which it is so frequently attributed. The period during which the patient had intermittent feverish attacks, was at least over seven years. Points that seem to call for special observation are:

1. The albuminuria which accompanied pregnancy, responded to treatment and cleared up after confinement in spite of the advancing kidney cystic changes.
2. The liver and kidneys apparently became involved about the same time, progress at first being more rapid in the kidneys.
3. There were never any symptoms of renal inefficiency or uræmia, the liver condition and not the renal apparently being that which led to a fatal result.
4. A period of remission in 1922, when the patient gained weight and looked remarkably well.
5. An impression was certainly gained that the cysts after formation may subsequently diminish in size and cystic formation may constantly recur and progress throughout the organ.



FIGURE SHOWING LIVER AND KIDNEYS DESCRIBED BY DR HOLMES.



# A RETRO-PERITONEAL HÆMATOMA IN HÆMORRHAGIC DISEASE OF THE NEW BORN.

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Paddington, Sydney.

THE following case is a rare form of hæmorrhagic disease and therefore worth reporting.

N.R., a female, aged three days, was admitted to the Royal Alexandra Hospital for Children on October 24, 1924.

She was perfectly healthy at birth and remained so until the third day, when she became pale and restless. The abdomen gradually became distended and the infant commenced vomiting. The bowels which had been open several times a day, became completely obstructed. The whole of the abdominal cavity appeared to be occupied by a hard, tender and immovable mass. The child on examination was found to be intensely anæmic. There was no evidence of blood either in the vomit or in the mucus which was returned after the bowel was washed out. There was no bleeding from the umbilicus nor were any petechial hæmorrhage present.

## Treatment.

A small incision was made in the middle line and the abdomen opened. There was no free blood in the abdominal cavity, although before the peritoneum was opened, some dark blood which had evidently tracked round from behind, was observed. The mass which could be felt before operation, was found to be a large retro-peritoneal hæmatoma, which had stripped the peritoneum from the whole of the posterior wall of the abdomen and had made its way between the layers of the mesentery and mesocolon, until in the latter case it had completely surrounded the ascending colon. It had also stripped a considerable portion of the peritoneum from the anterior abdominal wall. As soon as the cause of the trouble was seen, the abdomen was closed and ten cubic centimetres of healthy blood were injected subcutaneously, but the child died several hours later.

## Comment.

The infant was sent into hospital with the diagnosis of a possible intussusception. The shape, size and immobility of the mass, the persistent anæmia, the absence of blood in the fluid returned after a rectal injection and the age of the infant, were all points against such a diagnosis. The commonest form of bleeding in these cases is from the stomach or bowel. Occasionally the bleeding comes from the umbilicus and rarely there is bleeding under the skin or in the scalp, producing in the latter case a localized swelling resembling a cephal-hæmatoma. Occasionally hæmorrhage occurs into the peritoneal cavity or into the meninges. In this case the source of the bleeding was difficult to determine. The supra-renal capsules were examined *post mortem*, since hæmorrhage in these organs is not uncommon in these cases, but there was no hæmorrhage in the interior of the gland substance.

## Reviews.

### OPHTHALMOLOGY.

No introduction is needed to the excellent handbook on "Diseases of the Eye," by G. E. de Schweinitz.<sup>1</sup> The tenth edition is more imposing than its predecessors and its contents prove the painstaking endeavour of the author to bring his subject right up to date. In conformity with the modern tendency to elaborate methods of diagnosis and invent ever new instruments of observation, the earlier chapters give short accounts of Gullstrand's slit lamp,

<sup>1</sup> "Diseases of the Eye: A Handbook of Ophthalmic Practice for Students and Practitioners," by George E. de Schweinitz, M.D., LL.D. (Univ. of Penna.), Sc.D. (Univ. of Mich.); Tenth Edition, Re-set; 1924. Philadelphia and London: W. B. Saunders Company. Melbourne: James Little, Royal 8vo., pp. 865, with 434 illustrations and seven coloured plates. Price: 50s. net.

ophthalmoscopy with red-free light, contact illumination and other niceties of diagnosis. In Chapter VI. on conjunctivitis, there is described a new variety, agricultural conjunctivitis, of uncertain bacterial origin. The author should be supported in insisting on the instillation of 1% silver nitrate solution into eyes of the new born when even the suspicion of gonorrhœa cannot be excluded. The harvest of blind eyes from this field is a disgrace to the medical profession. Among diseases of the cornea, the striate clearing of corneal opacities described by Holmes Spicer is noted and illustrated. In the treatment of interstitial keratitis of congenital syphilis the author believes that "Salvarsan" in conjunction with mercury shortens the duration of the disease. The chapter on iritis any sympathetic ophthalmitis is worthy of careful reading; mention is made of the biological reaction to uveal tissue as an aid to diagnosis and slit-lamp examination of the endangered eye is suggested.

While recognizing the value of miotics and systemic treatment, such as strychnine and nitro-glycerine, in glaucoma, the author favours operative treatment. He advocates iridectomy in the acute form and gives a cautious preference for the filtering operations in the chronic disease. Among the operations described in Chapter XXI. is an ingenious one for ptosis, modified by J. O. Tansley on Hunt's operation. Besides the usual operation for cataract extraction, there are descriptions of the various intra-capsular methods, Smith's, Barraquer's and Arnold Knapp's. The author prefers the capsulotomy operation and, it is interesting to note, has given up simple extraction in favour of that with iridectomy. Reese's muscle-resection operation for squint is said to be popular in America and has much to recommend it. Sweet has written the account of his localization of foreign bodies in the eye. With certain exceptions, the posterior route is advocated in removing particles from the eye with the Haab magnet. His omission to mention the Mellinger, a much better magnet, is the worst word we can say against this admirable text-book.

### PERI-DUODENITIS.

PIERRE DUVAL, JEAN-CHARLES ROUX AND HENRI BÉCLÈRE in "Études Médico-Radio-Chirurgicales Sur Le Duodenum" draw attention to the frequency of duodenal deformity seen during radiographic examination in the presence of gall-stones.<sup>1</sup> In gall-bladder disease the duodenum is deformed, but the irregularity is not constant; in serial plates it is found that the duodenal "cap" always appears irregular, but the irregularity varies in the individual skiagrams. The authors attach great importance to this variation of the deformity in gall-bladder disease and state that it is one of the main distinguishing features from duodenal ulcer in which the deformity is constant in serial radiograms.

The pressure deformity of a distended gall-bladder on the duodenum is also regarded as an important factor in diagnosis, but they draw attention to the possibility of error owing to pressure from a gas distended hepatic flexure of the colon. The authors attach no importance to the filling of the ampulla of Vater as a factor in diagnosis of gall-bladder disease.

They state that after cholecystectomy on radiographic examination the duodenal "cap" invariably appears to be deformed and that this deformity is due to a "peri-duodenitis" which only causes symptoms if adhesions follow and cause some degree of stenosis. If stenosis occurs, gastro-enterostomy is recommended with occlusion of the pylorus. The occlusion is necessary to prevent food from passing by the pylorus with consequent irritation, causing the peri-duodenitis to persist.

If a "peri-duodenitis" exists before cholecystectomy, there is a great probability that it may persist even after the operation. The existence of gall-stones with severe gastric disturbance should suggest an accompanying peri-duodenitis, with the consequent *lendemain douloureux* after cholecystectomy. Peri-duodenitis may occur unaccompanied by other visceral disease and may even cause adhesions giving rise to a complete stenosis.

<sup>1</sup> "Études Médico-Radio-Chirurgicales Sur Le Duodenum," par Pierre Duval, Jean-Charles Roux et Henri Béclère; 1924. Paris: Masson et Cie; Demy 8vo., pp. 270, illustrated. Price: Frs 35, net.

## The Medical Journal of Australia

SATURDAY, FEBRUARY 14, 1925.

### Preservatives in Food.

THE regulation of a healthy and safe food supply would be a simple matter if the hygienic considerations were the only ones involved. Unfortunately, the wise counsel of the medical expert of playing for safety and of advocating the prohibition of the sale of food stuffs that contain foreign matter or that do not conform with arbitrarily established standards, often embarrasses the trade to a degree approaching impracticability. It must be remembered that the supply of common foods, such as milk, dairy produce, meat and fruit, is complicated by many difficulties. These foods are perishable; they cannot be produced in sufficient quantities where they are consumed; the producer and the vendor must be able to make a reasonable profit out of the traffic. Vested interests cannot be allowed to weigh when the public weal is at stake. Several months ago the Minister of Health of Great Britain appointed a committee to inquire into the use of preservatives and colouring matter in food. The inquiry has a wider significance than its reference would at first sight indicate. England, Scotland, Ireland and Wales are largely dependent on importations for various common articles of diet. Dairy produce, jams, other preserved fruits and vegetables, meat and some other substances are sent from Australia and New Zealand to the old country as well as wheat and sugar. The action taken on the recommendation of the committee in England will, therefore, have a profound commercial and economic effect in this country. In addition the decisions in Great Britain cannot fail to influence legislators here and thus the findings of the committee may exercise an indirect effect on the control of the food supply in Australia. The final report of the committee was issued late in October. It has recently been stated that no regulations will be introduced until those interested in the trade

and others interested in the hygienic aspect of the question have an opportunity of laying their views on the proposed alterations before the Ministry. In effect the report of the committee embraces a recommendation to prohibit the use of preservatives, although benzoic acid and sulphurous acid may be employed in coffee extract, in wines and cordials, in mineral waters, in sausages, in jam, in dried fruit, in preserved fruit and in beer and cider. The Committee advise a strict limitation to the amount of benzoic acid or of sulphur dioxide to be allowed for each specific purpose. Boric acid and salicylic acid are condemned in all circumstances, as are the even more harmful formaldehyde and fluorides.

The proscription of boric acid and salicylic acid as preservatives in butter, cream and other dairy produce is both reasonable and advisable. In the first place boric acid is believed to exercise a harmful influence on the human body. This action may be slight, but, if the statement made by the committee be correct, that the amounts used habitually are sufficient to supply the body with a continuous dose of boric acid, eventually a deleterious effect is likely to arise. In regard to salicylic acid the physiological action is more evident. It has been proved that neither substance is essential. Butter and cream can be packed in such a manner that they will remain in a good condition without artificial aid.

Of great interest to the Australasian meat industry is the recommendation that while salt, saltpetre, sugar, vinegar, acetic acid, alcohol and spices are not regarded as foreign preservative substances, sulphur dioxide is condemned. It is admitted that sulphur dioxide has not been shown to exert any harmful action in the concentration employed for this purpose. The argument used against its admission is that it may mask unsound meat and restore the red colour to meat that is already undergoing putrefaction. The members of the committee sought information concerning the chemical and biological action of sulphur dioxide on meat and endeavoured to discover some other agent which might assist the sulphites in preserving meat. No such substance has yet been discovered

in meat preserved in this manner in Australia, nor have these inquiries resulted in the disclosure of any masked putrefaction. Adequate inspection would suffice to detect whether or not the meat preserved by means of sulphur dioxide is always in a fresh and sound condition. It seems to us that the arguments used for the prohibition of this form of preservative in meat are insufficient and that for economic reasons all that is necessary would be to restrict the amount used. Freezing and cold storage is expensive and not always applicable and it is doubtful whether frozen meat is better or more wholesome than sulphur preserved meat.

With this exception the recommendations of the committee appear to us to be wise and capable of being adopted without hardship on the consumer, the producer or the vendor. If the prohibition of boric acid and salicylic acid is enforced in Great Britain, the effect will be watched with interest during the next two years. By that time there should be evidence of the advantage derived from this prohibition.

### Current Comment.

#### POLYCYSTIC DISEASE OF THE KIDNEY.

POLYCYSTIC disease of the kidney occurs in two forms. It is seen as a congenital manifestation in the newly born and it occurs in adults generally between the ages of forty and fifty. It attacks both sexes and may have a familial incidence. Sprent gave details of several cases in adults of the same family in the pages of this journal early in 1924. The case reported in this issue by Dr. H. I. Holmes is an interesting example of the adult type. Five cases of the same variety have recently been described by Mr. Roy F. Young.<sup>1</sup> The underlying cause of the formation of these cysts has not been determined and it may be of interest to review the various hypotheses which have been advanced to explain their occurrence.

The views expounded by Virchow were in effect that the cause was a sclerosis. He held that there was an inflammatory process in the kidney of the fœtus and that this resulted in obliteration of the communication between the collecting tubules and the pelvis. Many observers have since pointed out that the inflammatory process is the result and not the cause of the cyst formation. It has also been pointed out that these views cannot explain the epithelial proliferations which are some-

times found in the cysts. No epithelial proliferation was found in the kidney from Dr. Holmes's patient. Mr. Young also states that Virchow's views cannot explain the developmental defects which are met with apart from the lesions in the kidneys.

Another hypothesis is that the cysts are of the nature of new growths. This was first suggested by Malassez in 1877. It was supported by Brigidi and Severi in 1880. They concluded that the cyst contents were nothing else than the protoplasm of the epithelial cells fused together. They thought that the kidney may at first be affected by a formative irritation of the cellular epithelium of the uriniferous tubules and that succeeding this degeneration of the newly formed structures takes place. Nauwerck and Hufschmid supported this view and quoted instances in which there was evidence of new growth. Ritchie at about the same time expressed the opinion that the condition was one of new growth. He called attention to the budding processes in the early stage of cyst formation. He thought that the disease in childhood and that in adults were entirely distinct. He suggested that the adult cystic kidney should be classed with the adenomata and that the congenital variety should be regarded as being due to an error in development. In discussing the neoplastic hypothesis, Mr. Young refers to the work of Busse, whose finding of epithelial changes in all embryonal kidneys must be regarded as opposed to this view.

A third explanation offered for the origin of kidney cysts is that they are due to an error in development. This hypothesis is regarded by many as being the most admissible of all. It was put forward by Kuster, Ebstein, Rindfleisch and others. The collecting tubules of the kidneys are developed as outgrowths from the pelvis and ureter. The uriniferous tubules, on the other hand, are developed from the mesodermal metanephros. Connexions are normally formed between these structures. It is held that failure to establish these connexions results in the formation of cysts. The cysts often contain traces of urinary salts and it is thus maintained that retention of urine is a factor in their production. Obstruction of the uriniferous tubules alone will not cause a cyst; the proliferation of epithelium plays a rôle in their formation. Supporters of the developmental hypothesis then have to admit that the developmental error results in the obliteration of the uriniferous tubules and a proliferation of epithelium. Albarran and Imbert in discussing the infantile form of cyst formation, put this view very clearly. They said that it appeared to them that congenital cystic disease was the result of an error in development which was little understood, but which consisted essentially in obliteration of the uriniferous tubules situated more or less far from their communication with the calyces. This resulted in retention which by combination with an active process of proliferation led to the formation of cysts. The resemblance of cyst formation as seen in adults to that seen in the newly born and the frequent familial character of the disease have been regarded as favour-

<sup>1</sup> *The British Journal of Surgery*, October, 1924.



able to the acceptance of the developmental error hypothesis for the adult type. Many investigators, however, will ask why there should be such a length of time before the condition becomes manifest in an adult. Albarran and Imbert reply to this objection that many neoplastic conditions remain for years in a quiescent or dormant state.

Some workers in this field have tried to combine the various hypotheses. Thus Morris held that in all cases there was obstruction which resulted in dilatation. He thought that sometimes obstruction might be caused by fibrous proliferation around the tubules, sometimes it might be due to exudation into the tubules of some material which blocks them, in other cases again the tubules might become functionally isolated as the result of cicatrization after nephritis or of a congenital defect due to "the failure of union of the secreting with the carrying off parts of the kidney."

Mr. Young does not actually say which view he holds, but he describes the mal-development hypothesis as "most attractive." Dr. Holmes inclines to the view expressed by Cleland that some filtrable virus might be the causative factor. Cleland pointed out that the liver and the kidney are not related and he could offer no explanation why a developmental error occurring in one could so frequently be associated with a similar error in the other.

In considering the condition it must be conceded that Cleland's contention in regard to the simultaneous involvement of kidney and liver, as in Dr. Holmes's patient, is most pertinent. Those who hold the mal-development view, will find some difficulty in offering a satisfactory explanation of this dual condition. If some common cause could be discovered which was capable of producing failure or error in development in distinctly different organs, the problem would be simplified. In the absence of such a cause the explanation must be sought elsewhere. At the same time it is quite obvious that the postulation of a filter-passing virus is purely speculative.

#### ABNORMAL AND DIFFICULT LABOUR.

THE great army of general practitioners and the many specialists who are continually being confronted by the problems associated with abnormal and difficult labour will welcome the appearance of the first of the extra numbers of *The Lancet* which deals with these subjects.<sup>1</sup> The publication consists in a series of excellent monographs written by acknowledged authorities on obstetrics. In this way the book becomes to all intents and purposes a miniature system of practical midwifery.

Comyns Berkeley and T. Izod Bennett contribute a chapter on the toxæmias of pregnancy. They deal with vomiting of pregnancy, albuminuria during pregnancy and acute yellow atrophy of the liver. They do not discuss the pre-eclamptic state or eclampsia. This is done in a separate chapter by Gibbon FitzGibbon, the Master of Rotunda Hospital.

<sup>1</sup> *The Lancet* Extra Numbers, Number I.: Modern Methods in Abnormal and Difficult Labour.

Margaret Rorke describes purulent discharges and sores of the genitals which may occur during pregnancy and Lady Barrett discusses varicose veins in pregnancy. These two chapters are well illustrated.

T. Watts Eden deals with the indications for the induction of premature labour. He does not discuss all the conditions which may call for the induction of labour, but confines his attention to the more important conditions, such as toxæmic albuminuria, *ante-partum* hæmorrhage, *placenta prævia*, heart disease and pelvic contraction. He does not describe the clinical features and diagnosis of these conditions, but refers the reader to the chapters devoted to them. In this way Sir Squire Sprigge, the editor, has shown his wisdom, for much overlapping which might easily have occurred in such a composite work, has been avoided.

To J. P. Hedley has been entrusted the chapter on abortion and threatened abortion. He describes the clinical varieties, the signs and symptoms and diagnosis of abortion. In the treatment of incomplete abortion he holds that the finger and ovum forceps should be used. The curette should be used only when finger and forceps have failed and only then when there is no evidence of infection. In regard to the treatment of septic abortion he lays great stress on the necessity for the most careful and gentle treatment in emptying the uterus. The curette should not be used on any account.

H. Beckwith Whitehouse deals in an able and well illustrated chapter with *placenta prævia* and *ante-partum* hæmorrhage. He emphasizes the great difference between central and other varieties of *placenta prævia* both in regard to prognosis and treatment. He expresses the opinion that Cæsarean section will eventually have a very real and useful application in central *placenta prævia*.

Other chapters include: "Bony Deformities of the Pelvis," by J. M. Munro Kerr; "Occipito-Posterior, Face and Brow Presentations," by W. Gilliatt; "Breech Presentations," by Eardley L. Holland; "Transverse and Complicated Presentations," by Sir Ewen Maclean; "Multiple Pregnancy: Twin Labour," by J. Bright Banister; "Delayed Labour and Inertia," by J. S. Fairbairn; "Rigid Cervix and Tonic Contraction," by T. G. Stevens; "Post-Partum Hæmorrhage and Retained Placenta," by Sir George Blacker. In the chapter on injury to the birth canal and the displacements which may result from it, Victor Bonney has much to say in regard to the use of forceps, which should be read by every practising obstetrician. A. L. Flemming writes on anæsthesia in obstetrics and Miss M. Olive Haydon contributes an interesting chapter on anomalies and complications from the point of view of the midwife.

This book is a magnificent *résumé* of present day knowledge of a difficult subject. It is such a useful guide to treatment that it should be in the hands of every medical practitioner engaged in midwifery. In conclusion we should like to congratulate the editor on the new departure which has been made by his journal and on the admirable way in which the first of the extra numbers has been produced.

## Abstracts from Current Medical Literature.

### PHYSIOLOGY.

#### Measurement of Colour Blindness.

In all scientific investigations it is of importance to be able to express the results in numbers. In the case of colour blindness qualitative statements are usually made as to the nature of the defect or measurements in terms of arbitrary units. H. E. Roaf (*Quarterly Journal of Experimental Physiology*, April, 1924) has devised a method for the evaluation of colour blindness in terms of wave lengths. The method is based on the following premises. If a person be given coloured objects to match and colours are put together which to normal persons do not match, it should be possible to ascertain why the two individuals differ in their visual judgements. If the defect be purely one of the power of discrimination, mistakes ought to be found in any part of the spectrum. If, on the other hand, the defect be due to some lack of recognition of some physical quality of the coloured light, the defect should show some relation to the wave length of the light which is misjudged. This is not the same problem as the detection of colour blind individuals for protection from accidents. An outline diagram was made and coloured by water colour paints. An individual known to be colour blind was given a similar outline and the same box of paints. He was told to test the colours on a piece of white paper and to fill in the diagram to correspond with that given to him. The coloured diagrams were analysed by examining them in the light of a lantern from which any part of the spectrum could be excluded and the remainder of the spectrum recombined to give the complementary colour to that excluded. As the result of an examination of twenty-eight persons affected by colour blindness, it was found that the defect was always in the red end of the spectrum. The persons differed in the degree to which they failed to recognize differences in the red end of the spectrum. In some there was a correspondence to a cutting off of the spectrum to about  $\lambda$  6,200, others required the spectrum to be cut off at about  $\lambda$  5,800 and others required a reduction of about  $\lambda$  4,800 before the diagram matched the one from which it was copied.

#### The Active Principle of the Pituitary.

J. J. ABEL in the Harvey Lecture (*Bulletin of the Johns Hopkins Hospital*, October, 1924) discusses the physiological properties of the posterior lobe of the pituitary body and gives details of the separation, chemical and physiological properties of an extremely active preparation he

has made from the posterior lobe. He has separated three substances from the posterior lobe, a pressor substance in the form of a highly active tartrate and two depressor substances B and C, of which C has been shown to be histamine. Abel believes that the posterior lobe and *pars intermedia* contain but one specific principle possessing manifold physiological properties and that the depressor substances are not characteristic of this part of the pituitary. The author states that the position he has taken up, is diametrically opposed to that of certain other investigators who assume that at least three or even four or more specific hormonal principles exist in the extracts of the posterior lobe. In a properly made extract of the posterior lobe the powerful A substance entirely overbalances the action of the two other substances which are always present to a small extent. The active principle has been extracted in the form of a tartrate which is extremely active. Abel does not claim that this substance represents a single chemical individual, but impurities, if present, must be in very small amount. The activity of the tartrate has been compared with that of histamine phosphate on the guinea pig uterus. The most powerful preparations had an oxytocic value from 1,000 to 1,250 times that of histamine acid phosphate. A solution of the tartrate of 1 in 250,000,000 in Tyrode's solution produced as large a contraction of the guinea pig uterus as a 1 in 100,000 solution of histamine acid phosphate. With a more sensitive uterus, such as was used in some of the other experiments, probably a 1 in 75,000,000,000 solution of the tartrate would have produced an equally strong contraction. A dose of 0.01 milligramme raised the arterial blood pressure to an appreciable degree in cats and a dose of 0.05 milligramme or less caused a considerable diuresis in rabbits. It also affected in a characteristic manner the respiration both of the dog and rabbit, more especially in the unanaesthetized animal. Abel and Geiling demonstrated that there is in the grey matter of the floor of the third ventricle a tissue which yields an extract possessed of pressor and oxytocic properties or in other words, a tissue capable of elaborating a principle which appears to be identical with that found in the *pars posterior* and *pars intermedia* of the pituitary. In all so-called total hypophysectomies which have been hitherto performed in adult animals and which were followed by no observable symptoms, the amount of hormone producing tissue, although constituting only a small fraction of the total posterior lobe material, seems nevertheless to have sufficed for the production of the necessary physiological minimum quantity of an anti-diabetic substance.

#### Pulse Wave Velocity.

PULSE wave velocity, as measured by the hot wire sphygmograph has been shown to vary with the pressure

within the artery and with the extensibility of the arterial wall in experiments performed upon the excised carotid artery from animals and the human subject. Sylvia K. Hickson and B. A. McSwiney (*Journal of Physiology*, October, 1924) have studied the effect of variations in blood pressure on pulse wave velocity in the brachial artery of man. When a fall in blood pressure occurred on raising the arm to a vertical position, a lengthening of the carotid-radial time interval was observed, with an appreciable slowing of the pulse wave velocity. This postural variation in blood pressure is considerable and must be accounted for when the pulse wave velocity in arteries such as the femoral is estimated. In timing the arrival of the pulse wave at the wrist, asynchrony may be found if there is a difference between the position of the arms, the fact being of importance in certain clinical conditions, such as aneurysm of the arch of the aorta. Thus asynchrony of the two pulses in cases in which the external conditions are identical, indicates alteration in pressure in one artery, with delayed transmission of the pulse wave. These variations in pulse wave velocity with small changes in blood pressure are important as indications that constant modification of blood pressure is occurring independently of permanent alteration of arterial elasticity.

#### Vitiation of Air by Respiration.

T. B. SHAW AND W. I. GERRARD (*Journal of Royal Naval Medical Service*, October, 1924) have made observations on the vitiation of the air by respiration in submarines and its physiological effects on the personnel. While the observations bring out little that is new, they are held to confirm in a very definite way modern views on this subject. The submarine containing its normal complement was closed down as before diving, so that the entrance of air was entirely prevented. The available air space was approximately one hundred and seven cubic metres (three hundred and fifty cubic feet) per head. The air was kept in motion by table fans and in the first trial measures were used to remove the moisture and carbon dioxide added to the air by the personnel. The physical conditions of the air were thus kept very satisfactory during the trial. Sixteen men remained in the closed submarine for twenty-two and three quarter hours. At the end of twenty-two hours the carbon dioxide present was 0.94%; the oxygen was 16.37%. No complaints were made by any of the crew or observers, all of whom remained free from objective or subjective symptoms. The systolic and diastolic pressures, the pulse rate and respiration rate were determined at intervals. At the end of twenty-two hours the average systolic pressure showed a fall of 5.4 millimetres of mercury and the average pulse pressure a fall of 5.6 millimetres. These changes are very slight and cannot be attributed to

either the increase in carbon dioxide or the diminution of oxygen. In the second trial no measures were employed to remove carbon dioxide and moisture for the first twelve hours. After eleven hours the oxygen percentage was 18.72 and at the end of twelve hours the percentage of carbon dioxide was 2.27. Some discomfort was felt at the end of eight hours and at the twelfth hour each of the three observers complained of lassitude, headache, slight nausea and loss of intellectual power. Measures were then taken to remove moisture and carbon dioxide. At the end of the twenty-fourth hour there was 2% of carbon dioxide and 16.12% oxygen. Very slight physiological effects were noted towards the eighteenth and twenty-fourth hours. At no time during the trials was there any but the slightest rise in the rate of respiration and the percentage of carbon dioxide in the alveolar air was unaltered.

## BIOLOGICAL CHEMISTRY.

### Tomato Juice.

E. M. DELF (*Biochemical Journal*, May, 1924) has measured the anti-scorbutic power of the English tomato, used raw and fresh, when bottled, when put up in cans and after conversion into canned *purée*. The minimal daily ration of raw fresh juice of English tomatoes protecting young guinea pigs from scurvy has been found to lie between 1.5 and 2.5 cubic centimetres daily. It is superior to that of locally grown South African tomato which must be used in doses of four cubic centimetres daily. It is, however, inferior to fresh orange or lemon juice. A sample of bottled tomatoes which had been heated to 87.5° C. for forty-five minutes and stored for six months, had lost two thirds of its original value. When tomatoes are canned, they are heated to boiling point for five minutes and rapidly cooled in water. They lose in this process about threequarters of their anti-scorbutic quality. On storage they do not lose much more in anti-scorbutic strength. Canned tomato *purée* made by simmering pulp at 90° C. for one to two hours, loses seven eighths of its anti-scorbutic strength.

### Solubility of Lead in Serum.

L. T. FAIRHALL has measured the solubility of various compounds of lead in blood serum (*The Journal of Biological Chemistry*, July, 1924). An excess of dry lead salt was added to fresh horse serum, which was agitated and maintained within 0.1° C. of 25° C. The solid was removed by allowing the liquid to stand and by removing the last traces with the centrifuge. Portions of the clear liquid were analysed in triplicate, the lead being converted to chromate and titrated with 0.005 normal sodium thiosulphate. The author has found that lead oxide is about thirty times

as soluble in blood serum as other lead salts. More than one gramme dissolves in one litre. The author suggests that the ready solubility of lead oxide allows lead to enter the system more quickly than it can be eliminated or deposited in bones as relatively harmless lead phosphate and that the large quantities circulating tend to produce severe lead poisoning.

### Bile Acids in Blood and Urine During Pregnancy.

H. KLEESATTEL (*Klinische Wochenschrift*, October 7, 1924) has employed Hay's sulphur test for bile acids in the urine during pregnancy. The nearer the end of pregnancy, the more strongly altered is the surface tension of the urine and the more distinct the reaction obtained. A reaction was obtained in 75% of those tested at the commencement of labour and in 95% of those tested immediately after labour. There is a rapid decrease during the puerperium until by the end of the first week no reaction is obtained. Investigation of the bile acids in the blood showed that the normal amount is exceeded at the third month and a gradual increase is noted until full term. A sudden fall followed by a rise then occurs during the puerperium. This may be due to the onset of lactation.

### Ammonia in Blood of Rabbits.

R. F. LOEB, D. W. ATCHLEY AND E. M. BENEDICT have estimated the amount of ammonia in the venous and arterial blood of rabbits in the hope of obtaining information of value in respect to the view that urinary ammonia is manufactured in the kidney (*The Journal of Biological Chemistry*, July, 1925). A comparison has been made between the amounts of ammonia in the blood taken from the renal vein and *vena cava* in the rabbit and the dog with the amount found in the arterial blood from the femoral artery in both animals. An estimation has been made of the quantity of ammonia excreted in the urine. The dog excretes a much larger proportion of its urinary nitrogen in the form of ammonia. The authors find that the ammoniacal content of the renal vein of the dog is many times greater than that of the arterial blood, whereas in rabbits, the ammoniacal contents of arterial and venous blood are practically the same. The authors consider that their experiments lend definite support to the theory that urinary ammonia is formed in the kidneys.

### The Analysis of Urine for Lead.

L. T. FAIRHALL (*The Journal of Biological Chemistry*, July, 1924) has endeavoured to separate the lead compounds from urine by entrainment with the earthy phosphates. Attempts to separate lead from urine by absorption on charcoal or by precipitation upon bone meal or permutite, have not been found successful, but it was found possible to throw down the lead compounds along with the

earthy phosphate. This process is named entrainment by the author. It is well known that the phosphates of the alkaline earth metals are all precipitated when urine is made strongly ammoniacal. Provided that the urine be fresh or well preserved, the slow precipitation of earthy phosphates in the cold is efficient in carrying down the whole of the lead compounds. If the urine be heated in the usual way to render the deposition of phosphates more rapid, some of the lead remains in solution in the urine. By this method the author has detected such small amounts of lead as 0.05 milligramme in 5,000 cubic centimetres of urine from patients suffering from lead poisoning.

### The Nature of Blood Sugar.

W. DENIS AND H. V. HUME (*The Journal of Biological Chemistry*, July, 1924) have failed to confirm some of the experimental evidence on which has been based the hypothesis that the sugar in normal blood is the highly reactive  $\gamma$  glucose and that in cases of severe diabetes the blood sugar is the relatively stable  $\alpha$ - $\beta$  form of glucose. So far the principal evidence advanced in favour of this hypothesis is that extracts of normal blood freed from proteins show a progressive downward rotation when examined with the polariscope over a period of three days and that these extracts show a diminishing power to oxidize potassium permanganate at the same time. On the other hand, extracts of blood taken from persons with severe diabetes show these phenomena either not at all or in a much lessened degree. It has also been noted that the sugar content of normal blood as determined by the polariscope is invariably lower than when the results are obtained by titration. The authors have been entirely unable to confirm the findings in respect to the power to decolorize solutions of permanganate of potash, as no decrease in reducing power occurred in any of their extracts. As regards polariscopic observations, they have found that the gradual fall in rotation of extracts from oxalated blood over a period of three days was coincident with the appearance of moulds in them. When sodium fluoride was substituted for oxalate as an anti-coagulant, the changes in rotation were not observed. Such changes in rotation did not occur in "artificial blood extracts" when treated with fluoride, though they could be seen in solution treated with oxalates. The authors find that higher readings for sugar are obtained by the titration methods than by polariscopic readings with blood extracts and note that a similar disagreement of results is observed by the two methods with "artificial blood extracts," made from glucose salts and nitrogenous extractives. They infer that their results make it unjustifiable to assume that the experimental evidence previously advanced furnishes proof of the existence of  $\gamma$  glucose in blood.



## British Medical Association News.

### SCIENTIFIC.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on October 16, 1924, at the B.M.A. Building, 30-34, Elizabeth Street, Sydney.

#### Arterio-Sclerosis and the Nervous System.

DR. JOHN MACPHERSON read a paper entitled: "Effects of Arterio-Sclerosis on the Nervous System and Their Treatment" (see page 153).

A paper written by DR. J. BOSTOCK and DR. H. M. NORTH entitled: "Arterio-Sclerosis and Mental Disease," was read by DR. G. L. EWAN (see page 156).

DR. S. EVAN JONES gave a verbal account of the analysis of the blood pressure and of the form of mental disease in six hundred patients of the Broughton Hall Psychiatric Clinic.

PROFESSOR A. E. MILLS congratulated Dr. Jones on his observations and pointed out that the emotional factor was of great importance in regard to isolated readings of blood pressure. It was frequently found that the blood pressure of a patient was five to fifteen millimetres of mercury lower at a second reading than at a first. In general, there was great confusion concerning the subject of arterio-sclerosis and it was important to distinguish the disease from atheroma. Arterio-sclerosis was a definite pathological state not to be included with atheroma. The latter condition was due to syphilis. Arterio-sclerosis meant the narrowing of the vessel which would lead to increase of blood pressure if the heart were efficient. Increase of blood pressure was not altogether dependent upon the heart, but rather was due to changes in the vessels. Increased blood pressure alone would not cause cerebral changes. Neurone destruction was due not to the blood pressure, but to the fact that certain vessels had become obliterated and malnutrition of cerebral tissue resulted.

Professor Mills criticized the classification of arterio-sclerosis adopted in the paper submitted by Dr. Bostock and Dr. North and maintained that the mere palpation of the vessel wall was not a sufficiently accurate basis on which to form a classification.

DR. S. J. MINOGUE read a paper entitled: "Hysterical Delirium" (see page 162).

DR. ANDREW DAVIDSON congratulated Dr. Minogue upon his paper.

DR. DONALD FRASER said that Frederic Myers had anticipated Freud in stating that hysteria was a disease of the subconscious. Patients suffering from hysteria were always in a state of "hypnosis." Their condition was the result of suggestion—auto-suggestion or hetero-suggestion. That was the reason why it was often very difficult to hypnotize these sufferers. They were already hypnotized. To understand hysteria they must thoroughly understand the psychology of hypnoidal states. Hypnosis might be defined as a heightened state of receptivity of suggestion. They would then have to define suggestion as a "successful appeal to the subconscious." Not all appeals penetrated to the depths of human personality. One would then have to regard hysteria as a hyper-sensitiveness to suggestion. It might be the sufferer's own or auto-suggestion, or the suggestion of another, very often his physician. The treatment lay in curing the hyper-sensitiveness and eliminating the causal suggestion by some other.

DR. EVAN JONES said that there was often difficulty in treating hysterical patients by means of hypnosis, but emphasized the importance of the social factors in the causation and made plea for easier access to the divorce court for many such patients.

DR. RALPH NOBLE referred to the doctrine of the late W. H. R. Rivers, who taught that hysteria was always the result of conflict in the subconscious. There was always a subconscious motive for hysterical symptoms, as in the hysterical paralysis of war neuroses. Both Rivers

and Professor Elliott Smith by their ethnological work, had thrown considerable doubt upon the theories of Freud and Jung regarding symbolism as expressed in dreams. He felt that frequently Freud and his followers saw the solution to the problem confronting the patient and unconsciously directed the analysis in such a way that their solution was reached. During such a process suggestion must play an important part.

DR. R. WINN emphasized the importance of Freud's use of free association, which he asserted was a scientific method.

DR. S. J. MINOGUE in reply stated that the immediate cause of hysteria was loss of control by the patient. Frequently hysteria appeared in patients, predisposed to the condition, who possessed a psychopathic personality. The social factors were of the greatest importance.

### NOMINATIONS AND ELECTIONS.

THE undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Goldberg, Solomon, M.B., Ch.M., 1924 (Univ. Sydney), 82 Ashley Street, Chatswood.

Hamilton, Marie Montgomerie, M.B., Ch.M., 1923 (Univ. Sydney), "Tomabil," Wentworth Road, Strathfield.

Stayner, Frederick Eastwood, M.B., Ch.M., 1924 (Univ. Sydney), Rowe Street, Eastwood.

Amphlett, Julie Leonie, M.B., Ch.M., 1923 (Univ. Sydney), 28 Crow's Nest Road, North Sydney.

The undermentioned have been nominated for election as members of the Victorian Branch of the British Medical Association:

Hallett, Claude Vincent, M.B., B.S., 1924 (Univ. Melbourne), Swan Street, Burnley.

Allen, Thomas Kingsley Russell, M.B., B.S., 1924 (Univ. Melbourne), 5, Levein Street, Essendon.

Dunn, Victor Henry Lavington, M.B., B.S., 1924 (Univ. Melbourne), 94, Ivanhoe Parade, Ivanhoe.

## Medical Societies.

### THE SYDNEY HOSPITAL CLINICAL SOCIETY.

A MEETING OF THE SYDNEY HOSPITAL CLINICAL SOCIETY was held in the Lecture Hall, Sydney Hospital, on September 25, 1924, DR. T. FLASCHI, D.S.O., the President, in the chair.

#### Diverticulitis of the Colon with Perforation.

DR. H. S. STACY related the clinical history of a patient, aged forty-two years, who had been admitted to hospital on September 9, 1924, with a history of the occurrence of sudden severe pain in the lower part of the abdomen twenty-four hours previously. Shortly after the onset of pain the bowels had moved three or four times. As far as was known the stools had contained no blood. After some time the pain had become easier, but it returned soon afterwards and continued up to the time of admission. The patient had not vomited and the past history had contained nothing of significance. Examination on admission had revealed that the patient lay in bed with his knees drawn up and with an anxious expression on his face. The tongue had been dry and furred. The abdomen had moved slightly on respiration, but definite rigidity had been present on both sides in its lower portion. There had been no distension. No definite dullness had been present in the flanks. General tenderness had been present. The liver dullness had been normal. The patient's mental condition had been clear. The most striking feature had been the feeble character of the pulse, the rate had been 110 in the minute. The temperature had been 37.9° C. (100.2° F.). The respiratory rate had been 42. The leucocytes had numbered 2,300 per

cubic millimetre. The feeble nature of the pulse had indicated that the patient was in a very serious condition and had shown that laparotomy offered the only hope of recovery. Beyond the fact that peritonitis was present, the diagnosis had been obscure. The abdomen had been opened in the middle line below the umbilicus. The peritoneal cavity had contained more than a litre of dark, offensive fluid of the consistency of soup. The intestine had been very congested and an old standing Lane's kink had been found near the termination of the ileum. The bulk of the fluid had been removed by pads and the abdomen had been closed without the primary lesion having been discovered. As a desperate hope, lymphaticostomy had been attempted, but without success, owing to the bad condition of the patient. Death had occurred about four hours afterwards.

Dr. Stacy pointed out that the most notable clinical feature and the one denoting the intense gravity of the lesion was the extremely soft pulse. This had made him realize that he was dealing with an ordinary condition such as acute appendicitis. Autopsy had revealed the cause of this in the cloudy swelling of the myocardium which was evidently toxic in origin. A similar soft pulse was seen in intestinal obstruction and in both conditions the good mental state of the patient might deceive the unwary.

*Post mortem* examination had revealed a dirty fluid of fecal odour and appearance in the pelvis and the left peritoneal gutter. No pus had been seen. The intestines had been normal with the exception of the descending colon, which for a length of about fifteen centimetres at the pelvic brim had been greatly hypertrophied and had appeared fibrotic. This condition had merged gradually into normal bowel above and below. In the lower half of the thickening below the main layer there had been a gangrenous diverticulum about the size of a terminal phalanx of the thumb. When pressure was made upon it, faeces had escaped from a small perforation on its summit. The diverticulum had been thin-walled, it had contained no fat in its summit and had been quite distinct from the *appendices epiploicae*. It had bulged on the free peritoneal surface. There had been no obstruction of the bowel either at the site of the hypertrophy or below. Dr. Keith Inglis had examined the specimen microscopically. He had said that sections of the thickened bowel wall near the diverticulum and at a distance revealed great thickness of the wall, which was mainly due to increase of involuntary muscle without much fibrosis. The muscle cells deviated somewhat from normal in that they seemed swollen and some of the nuclei were narrow and wavy. In the bowel wall near the diverticulum were accumulations of inflammatory cells (neutrophile leucocytes, eosinophile cells, lymphocytes and plasma cells); these were conspicuous and the serous surface of the bowel was the seat of recent inflammatory changes due to the infection of the peritoneal cavity. Dr. Inglis had added that he was inclined to think that the increased thickness of the bowel wall and the diverticulum had both been present for a long time and that the perforation of the diverticulum had led to the acute inflammatory changes in the bowel wall and in the peritoneal cavity.

Dr. Stacy added that definite pallor and cloudy swelling of the liver, kidneys and heart muscle had also been found.

In the discussion which followed, comment was made on the small size of the diverticulum and on the minute perforation which was barely perceptible *post mortem*. It was pointed out that recovery after the rupture of a diverticulum was extremely rare. The difficulty in diagnosis, the extreme collapse and the cloudy swelling of the organs all helped to make treatment unsatisfactory in the majority of instances.

#### Compound Fracture of the Tibia and Fibula.

Dr. Stacy also showed a man, aged eighty years, who had been admitted to hospital on August 20, 1924, after having been knocked down by a motor car. He had sustained a compound fracture of the left tibia and fibula in the lower third. A radiogram by Dr. Edwards had revealed backward, outward and upward displacement of

the lower fragment of the tibia with some comminution. About eighteen hours after admission the patient had been taken to the operating theatre and the wound had been completely excised and a few comminuted fragments of bone had been removed. It had been found that the backward and upward displacement was even more pronounced than was indicated by the skiagram. By means of Lane's instrument the bones had been manoeuvred into an end to end position. A Lane's plate with four screw holes had then been inserted, the wound had been closed and the leg had been put up in back and side splints. Convalescence had been uneventful. After five days the leg had been put up in plaster of Paris and a few days later a window had been cut in the plaster. On the sixteenth day the stitches had been removed and the wound had healed by first intention. The following day the patient had been discharged from hospital on crutches. At the time of demonstration five weeks had elapsed since the operation. The leg was still in plaster and the patient would not be allowed to put any weight on the fractured limb for quite fourteen weeks from the date of operation.

Dr. Stacy pointed out that the interesting features of the case were several. In the first place the patient was an old man. In the second place pain had been relieved as a result of getting the bone into good alignment. Dr. Stacy had noticed this repeatedly after the open operation and plating. The third point was the quick time in which the patient had been able to leave the hospital (seventeen days). Fourthly, early excision of the wound had enabled him to get healing by first intention and in this way the success of the operation had been secured. Lastly, Dr. Stacy said that skiagrams taken from two aspects, although invaluable, did not always convey such exact information as the surgeon got with the naked eye in an open operation.

Opinions of those who discussed the case, were divided as to the advisability of plating compound fractures. It was suggested that the position of the fragments might not necessarily be improved, whereas there was a risk of setting up prolonged suppuration.

#### Hæmaturia.

Dr. Stacy also showed a man, aged fifty-six, who complained of the frequent passage of blood-stained urine. There had been no complaint of pain, but the patient had suffered from retention of urine. There was no palpable hypertrophy of the prostate gland and the renal efficiency tests revealed very poor kidney function. Cystoscopic examination was difficult on account of the large quantity of blood in the urine. A cystogram had been taken, but its interpretation was obscure. Dr. Stacy asked for advice as to the best procedure to be adopted in the determination of a diagnosis. Cystoscopic examination accompanied by a constant flushing of the bladder was suggested. It was thought that a median prostatic bar might give rise to the symptoms in this patient.

#### Hæmato-Myelia.

DR. LESLIE DUNLOP showed a middle aged male patient who had been thrown from a sulky on to his head. He had immediately become paralysed in all four limbs and there had been loss of control of the bladder and rectum soon after the accident. Control of the sphincters had been regained within a month and the patient had gradually improved during the twelve months that had elapsed since the fall. Dr. Dunlop pointed out that the patient was able to walk with a very stiff gait without the aid of crutches. He had good use of both arms, except that there was weakness and wasting of the small muscles of the right hand. There was no anaesthesia. All four limbs were spastic, the legs particularly so. The arm and leg reflexes were brisk and there was ankle clonus as well as patellar clonus. The plantar reflex in both lower limbs was extensor in type. The patient complained that his legs became drawn up when he was in bed; this was the spinal automatic reflex. X-ray examination had revealed no fracture or dislocation of the cervical vertebrae. Dr. Dunlop said that the history and the recovery were typical of hæmorrhage into the spinal cord (hæmato-myelia). The prognosis was moderately good.

During discussion it was suggested that much further improvement was unlikely to occur. Speakers agreed with the diagnosis and pointed out that a fairly early recovery with a residue of unilateral signs was characteristic of hæmato-myelia.

#### Syphilitic Spastic Paraplegia.

Dr. Dunlop also showed a patient suffering spastic paraplegia of gradual onset. The signs of this condition were characteristic, but the man had much better use of his legs than the preceding patient. Lumbar puncture had revealed a clear cerebro-spinal fluid containing an excess of lymphocytes. The fluid had reacted to the Wassermann test. A diagnosis of syphilitic meningo-myelitis had been made and the patient had been treated with iodides and intravenous injections of an arsenical preparation. No definite improvement had been noted.

Several speakers advocated intensive treatment with mercury and iodide of potassium.

#### Tumour of the Forehead.

DR. G. R. HAMILTON showed a woman, aged fifty-six years, with a tumour of the forehead of three years' duration. There had been a rodent ulcer affecting the nose four years previously. An operation had been performed and a flap had been taken from the forehead and brought down to cover the area left after excision of the ulcer. At the time of demonstration there was a rounded tumour the size of half a walnut in the middle of the forehead. The tumour was firm and appeared to move freely. X-ray examination had revealed erosion of the frontal bone. Dr. Hamilton thought that the condition was carcinomatous and suggested that a portion of the ulcer might have been included in the flap and that a change of type had subsequently taken place.

Several speakers considered that the condition was not necessarily malignant, but that in any event there might have been a spread upwards of the original growth from the root of the nose. An exploratory operation for the excision of a piece of the growth for pathological examination was advised.

#### Skiagrams.

DRS. J. G. AND W. A. EDWARDS showed a number of interesting skiagrams in addition to those relating to the patients who had been demonstrated. The conditions depicted in the skiagrams included single pneumothorax, spurs on the *os calcis*, osteo-chondroma of the radius, foreign body in the colon, dislocation of the astragalus. Characteristic pictures of ulcer and carcinoma *ventriculi* were also shown.

In one skiagram was shown a double pneumothorax. In this patient one lung had subsequently expanded in the comparatively short time of five days.

## Obituary.

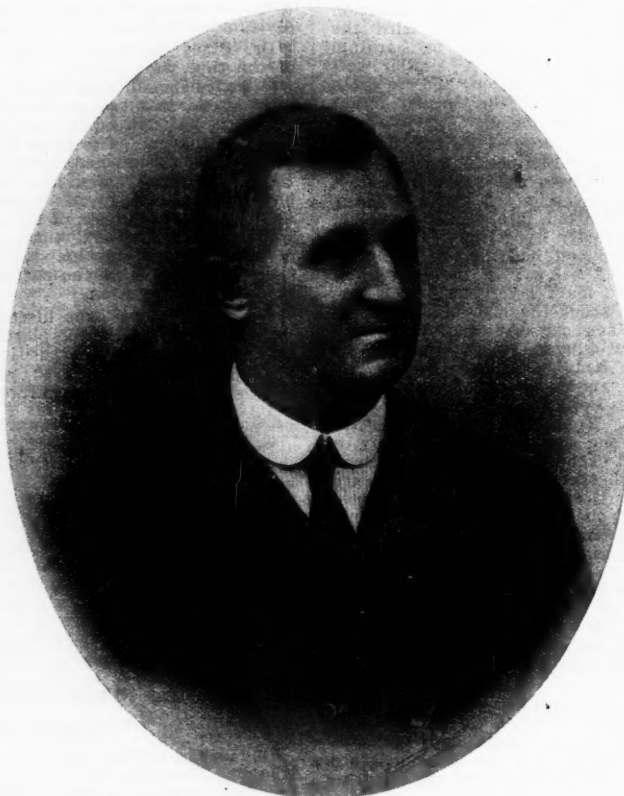
### SYDNEY JAMIESON.

In the world of science only a few can claim a permanent place. Many labour well, gain the approbation and esteem of their fellows and yet leave no lasting evidence of their good works. They are soon forgotten in this busy, hustling world. Sydney Jamieson has left his footprint on the road and it will be long e'er it is obliterated. His was a creative mind and what he initiated will not be thrust aside to crumble into nothingness.

Sydney Jamieson was born in London of a Scottish father and English mother just over sixty years ago. In 1871, when he was seven years of age, the family came to Australia. Sydney Jamieson attended Horton College, at Ross, for a time. Later he entered the Sydney Grammar

School. He displayed considerable ability at his school work and at the same time manifested prowess in the cricket field and on the football ground. From there he passed on to the Hurlstone College and matriculated at the age of seventeen. He went into residence at Saint Andrew's College. At the University of Sydney, as at school, he worked well and played well and gained great popularity among the men of his year. His teachers recognized that he was a man of considerable promise. He took the degree of Bachelor of Arts in 1884 and immediately left for the Old Country. He entered the medical school of the University of Edinburgh in 1884. His career as a medical student was an eminently successful one. His keenness, his natural ability and his originality of thought soon attracted attention. In 1888 he secured the degrees of bachelor of medicine and master of surgery. About this time he showed much aptitude and interest in pathology and seized the opportunity offered to him by his master, Professor Greenfield, to teach pathology as demonstrator. In the following year he passed the examinations of the Conjoint Board of the Royal College of Surgeons of England and the Royal College of Physicians of London. After spending some time in Edinburgh as a pathologist he went to Germany and studied for about six months under the late Robert Koch. He then returned to Sydney and started in practice as a physician.

Equipped with an unusually sound knowledge of pathology and a wide grasp of general medicine, he had no difficulty in establishing himself as a first-class practitioner. His ability was recognized at once, for he was appointed an honorary assistant physician at the Sydney Hospital within twelve months of his return to Australia. Pathology continued to hold his attention and his conviction that medical practice to be sound must be founded on unassailable pathological knowledge stimulated him to create facilities for laboratory practice. He realized the





need for a department of pathology at his hospital and succeeded in convincing the hospital authorities. The department was established through his energies and advocacy and was placed under his care. Shortly after he was appointed curator of the pathological museum at the University of Sydney, actually before a museum existed. He worked hard and with consummate skill. With the aid of the late Thomas Anderson Stuart, then Dean of the Faculty of Medicine and founder of the medical school, he gathered together a fine collection of specimens and arranged them in excellent order, so that as fresh material was added, the foundation could remain unaltered. In this work he displayed ingenuity, prevision and originality. Unlike many leading pathologists in other parts of the world, he did not allow a personal predilection for one special branch of pathology to restrict the completeness of his arrangement or to interfere with the teaching value of the museum.

In the course of relatively few years Sydney Jamieson was appointed full physician at the Sydney Hospital and later he became the senior honorary physician. The value of his services to the institution cannot be over-rated; they were splendid. His knowledge and judgement, his untiring energy, his love for his fellow men and his geniality combined to make him an ideal physician.

On the death of W. H. Goode in 1902 he was appointed lecturer on medical jurisprudence and toxicology at the University of Sydney. He held this office for five years. A little later he became consulting pathologist to the Sydney Hospital and the actual work in the laboratories was carried out by younger men. He was also lecturer on clinical medicine at the University. He conducted a considerable amount of research in his laboratory and proved by practical experience that a busy practitioner conducting a large private and hospital practice as a physician can find time to carry out pathological work.

When war broke out Sydney Jamieson did not hesitate to do his duty. In May, 1915, he embarked with a contingent of the Australian Army Medical Corps for Egypt. He served with the rank of lieutenant-colonel at Lemnos in the Number 3 Australian General Hospital. In this way he gave his whole energies and his fine ability to his country and displayed the same unselfish thoroughness that characterized the whole of his career. After his return to Australia in 1916 he continued to give valuable war service to the Australian Army Medical Corps at the Number 4 Australian General Hospital at Randwick, Sydney.

His knowledge of and interest in hygiene and preventive medicine proved of great value to the State Health authorities and to successive New South Wales governments. His advice was sought on many occasions in connexion with many matters affecting the public health. At times he took a very strong attitude concerning the prevention of disease and when the public weal or safety was at stake, he did not hesitate to rebuke the health authorities. He could be very severe with his pen. In 1914 he was chosen a member of the advisory committee to the Government of New South Wales on the subject of tuberculosis. During the influenza epidemic he together with several others sought without avail to guide the Government of the State in dealing with the situation.

He was for twenty-seven years honorary examining medical officer for the Queen Victoria Homes for Consumptives and for fourteen years he was an active member of the Board of Directors. For five years before the Thirlmere Home was taken over by the Executive of the Queen Victoria Homes, he acted as honorary examining medical officer for that institution. He advised the board when difficulties arose and visited the sanatoria both at Wentworth Falls and Thirlmere.

Reference has already been made to his popularity and the high esteem in which he was held by his colleagues and contemporaries. Sydney Jamieson was elected a member of the British Medical Association at the beginning of his professional life. Two years after his return to Sydney he was chosen by his fellow members to be a member of the Council of the New South Wales Branch of the Association. Each year from 1901 until his death he was re-elected. In 1912-1913 he was chosen to be

the Vice-President and in the following twelve months he was President. He was a very regular attendant at the meetings of the Branch and took part on very many occasions in the scientific discussions. He attended many of the Australasian Medical Congresses, including the first session of the reorganized congress in Melbourne in 1923 and contributed many valuable papers.

Sydney Jamieson was a charming companion, a real friend and an ideal husband and father. His patients loved him and were loud in their praise of his gentleness, attention and unselfishness. His generosity was lavish, impulsive and humane. He was prepared to share all he had with those he liked and to give away more than he could afford when his sense of pity was aroused. His genial smile was infectious and often disarmed his critics. His kindness to others often led him into difficulties, but he never regretted a good deed, even if others of a more cautious temperament regarded it as an unwise one. His friends have much reason to mourn their loss. The sympathy of the whole medical profession is extended to his wife, son and daughter.

#### JAMES MACKENZIE.

THE death has been announced by cable of Sir James Mackenzie, which took place in London on January 27, 1925. During the last few years age had begun to impress its mark on him. His great career may be said to have ended some years ago. He was born in 1853 and was educated in Perth, Scotland, and the University of Edinburgh. At an early age his brilliant intellect was manifested when, shortly after graduation, he served as assistant to the Professors of Clinical Medicine at the University of Edinburgh. He was a general practitioner at Burnley in Lancashire from 1878 to 1907. While still engaged in this busy life he turned his attention to several problems connected with cardiac lesions. Soon after Einthoven, who has just been honoured by the award of the Nobel Prize for Medicine, published the results of his first investigations of the cardiac function with the aid of his electrocardiograph, James Mackenzie conceived the principles on which modern cardiology are based. He realized that cardiac disease was in the majority of instances a disturbance of the nodal mechanism and not the result of an inflammatory lesion of valves interfering with the mechanical maintenance of the circulation. It is unnecessary to recall in this short note the part played by James Mackenzie in the building up of the doctrines of cardiology which have attained universal acceptance during the last decade or two. His name and that of Thomas Lewis stand out boldly in the records. His knighthood dates back to 1915. He was elected a Fellow of the Royal Society in 1911. Since that date honours have been showered upon him by the authorities at many British and foreign universities. His publications have commanded great attention not only in English-speaking countries, but also in other lands. Some of his books have been translated into many languages. He had a great personal charm and a commanding presence. James Mackenzie will figure as one of the most eminent practitioners of his time.

#### THE MELBOURNE UNIVERSITY VETERINARY RESEARCH INSTITUTE.

WE have been requested by Dr. Georgina Sweet, the Acting Director of the Melbourne University Veterinary Research Institute, to appeal to members of the medical profession for specimens of *Ascaris lumbricoides* that have been obtained from human beings. The parasites are required for the purposes of an investigation which is being carried out at the Institute and even single specimens will be welcomed. It is essential that specimens should be forwarded whole and immediately they are found to the Institute at Story Street, Parkville, Victoria. The parasites should be washed in normal saline solution and enclosed in a clean bottle without any surrounding fluid. No preservatives should be used.

## NOTICES.

A MEETING OF THE SECTION OF PSYCHIATRY AND NEUROLOGY OF THE VICTORIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION will be held on February 16, 1925, at the Walter and Eliza Hall Institute of Research in Pathology and Medicine. Professor W. A. Osborne will read a paper entitled: "The Purposeless Reflex and the Signal Reflex." Members of the Branch are invited to attend.

## Books Received.

- DER AUSFALL DES KOPFHAARS UND SEINE BEHANDLUNG, von Prof. Dr. med. Felix Pinkus; 1924. Halle a. S.: Carl Marhold Verlagsbuchhandlung; Demy 8vo., pp. 65. Price: 2 Marks, equal approximately 2s. 3d.
- ENFERGENE ENTSTEHUNG DER MAGEN-UND ZWÖLF-FINGERDARM-GESCHWÜRE, von Professor Dr. Adolph Posselt; 1924. Halle a. S.: Carl Marhold Verlagsbuchhandlung. Demy 8vo., pp. 55. Price: 1.80 Marks, equals approximately 2s.
- DIE BEZIEHUNGEN DER GENTALORGANE ZU HAUT-VERÄNDERUNGEN, von Dr. Kurt Wiener; 1924. Halle a. S.: Carl Marhold Verlagsbuchhandlung. Demy 8vo., pp. 77. Price: 2 Marks, equals approximately 2s. 3d.
- HANDBUCH DER BIOLOGISCHEN ARBEITSMETHODEN (Handbook of Biological Methods), Edited by Geheim-Medizinal-Rat Professor Dr. Emil Abderhalden, with collaboration of 500 Eminent Specialists; Division IV.: ANGEWANDTE CHEMISCHE UND PHYSIKALISCHE METHODEN (Applied Chemical and Physical Methods), METHODEN ZUR BESTIMMUNG DES GASWECHSELS BEI TIEREN UND MENSCHEN (Methods for the Determination of the Gaseous Metabolism in Animals and Men), by Francis G. Benedict, Carnegie Institution, Washington; 1924. Berlin: Urban und Schwarzenberg. Crown 4to., pp. 674. Price: 9.60 Marks, equals approximately 10s.
- HANDBUCH DER BIOLOGISCHEN ARBEITSMETHODEN (Handbook of Biological Methods), Edited by Geheim-Medizinal-Rat Professor Dr. Emil Abderhalden, with Collaboration of 500 Eminent Specialists; Division V.: METHODEN ZUM STUDIUM DER FUNKTIONEN DER EINZELNEN ORGANE DES TIERISCHEN ORGANISMUS (Methods for the Study of the Function of the Individual Organs of the Animal Organism); THEORETISCHE GRUNDLAGEN DER GELENKMECHANIK (The Theoretical Basis of the Mechanics of Joints), by R. Grammel; GRAPHISCHE METHODEN ZUR DARSTELLUNG NORMALER UND PATHOLOGISCHER WILLKÜRLICHER BEWEGUNGSABLAUFE (The Graphic Method of the Illustration of Normal and Pathological Voluntary Excursions of Movement), by E. Hirt; 1924. Berlin: Urban und Schwarzenberg. Crown 4to., pp. 390. Price: 5.40 Marks, equals approximately 6s.

## Medical Appointments.

Dr. Malcolm D. H. Harpur (B.M.A.) has been appointed Senior Resident Medical Officer, Fremantle Public Hospital, Western Australia.

Dr. C. R. Dunkely has been appointed Junior Resident Medical Officer, Fremantle Public Hospital, Western Australia.

Dr. Francis G. M. Simpson has been appointed District Medical Officer, Tambellup, Western Australia.

Dr. Ernest Edward Wilbe (B.M.A.) has been appointed Public Vaccinator at Manangatang, Victoria.

The undermentioned have been appointed Members of the Advisory Committee in connexion with Medical Inspection in State Schools, Victoria:

- Sir James William Barrett (B.M.A.).  
Dr. Bernard T. Zwar (B.M.A.).  
Dr. Edith Helen Barrett (B.M.A.).  
Dr. Edward Robertson (B.M.A.).  
Dr. Thomas Walker Sinclair (B.M.A.).

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser," page xviii.

CARCOAR DISTRICT HOSPITAL, N.S.W.: Resident Medical Officer.

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C.

| BRANCH.   | APPOINTMENTS.   |
|---|---|
|   | Australian Natives' Association.<br>Ashfield and District Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies' Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phoenix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.          | All Institutes or Medical Dispensaries.<br>Australian Prudential Association.<br>Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.  |
| QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane. | Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital.   |
| SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.            | Contract Practice Appointments at Renmark.<br>Contract Practice Appointments in South Australia.  |
| WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.        | All Contract Practice Appointments in Western Australia.  |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.            | Friendly Society Lodges, Wellington, New Zealand.   |

## Diary for the Month.

- FEB. 17.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
FEB. 17.—Tasmanian Branch, B.M.A.: Council.  
FEB. 17.—Illawarra Suburbs Medical Association, New South Wales.  
FEB. 20.—Central Southern Medical Association, New South Wales.  
FEB. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee: Organization and Science Committee.  
FEB. 25.—Victorian Branch, B.M.A.: Council.  
FEB. 25.—Section of Surgery, New South Wales Branch, B.M.A.  
FEB. 27.—Queensland Branch, B.M.A.: Council.  
MAR. 3.—New South Wales Branch, B.M.A.: Ethics Committee.  
MAR. 3.—Tasmanian Branch, B.M.A.: Council.  
MAR. 4.—Victorian Branch, B.M.A.: Branch.  
MAR. 4.—Section of Obstetrics and Gynaecology, New South Wales Branch, B.M.A.  
MAR. 6.—Queensland Branch, B.M.A.: Branch.  
MAR. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
MAR. 10.—Tasmanian Branch, B.M.A.: Branch.  
MAR. 12.—Victorian Branch, B.M.A.: Council.  
MAR. 12.—South Australian Branch, B.M.A.: Council.  
MAR. 13.—Queensland Branch, B.M.A.: Council.

## Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone B. 4635.)

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